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JOURNAL OF FORENSIC SCIENCES

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The Journal of Forensic Sciences is the official publication of the American Academy of Forensic Sciences. It is devoted to the publication of original investigations and observations in the various branches of the forensic sciences; these include forensic pathology, toxicology, psychiatry, immunology, jurisprudence, criminalistics and questioned documents. The journal will be published quarterly, one volume of four issues appearing each year.

LOUIS J. REGAN, M.D., LL.B.
1892–1955

The sudden and unexpected death of Doctor Louis J. Regan brought real sadness to his many devoted friends, his colleagues and his family. His devotion and his untiring efforts in the fields of Medicine and Law, his investigations and administration in the educational field of the Forensic Sciences, gave him a commanding position in his profession. He was learned in the arts and literature, a meticulous teacher who was able to impart the zest for learning to his students and colleagues. Above all, his integrity, honesty and humaneness inspired everyone associated with him to constantly search for the good in man.

Dr. Louis J. Regan, the fourth President of the American Academy of Forensic Sciences, was born in East Bloomfield, New York, February 20, 1892. He received his medical education from George Washington University Medical School,

where he graduated in 1913. The following years he saw wide service in various hospitals, including postgraduate courses in Paris, Berlin, and at the Chicago Postgraduate Medical School.

In 1916 he began his war service and was Chief of Surgery at Columbus Barracks, Ohio, and at the General Hospital No. 12 at Asheville, North Carolina; then in the Department Hospital, U. S. A., Manila, P. I. He also studied at the Mayo Clinic, on leave for that purpose during the war. Dr. Regan was later appointed Chief Surgeon of the Chinese Expedition and was stationed at Tientsin, returning from there to San Antonio, where he retired in 1921, as a major. Shortly thereafter he entered the practice of medicine in Los Angeles as a Specialist in Obstetrics and Gynecology.

Sometime later he suffered an injury to his spine and was bedridden for a long period. During that time he read many books pertaining to law, and his study was of such an intense nature as to qualify him for a degree in law which he received in 1938 and was then admitted to the California State Bar. Because of his profound interest in the medical and legal professions, Dr. Regan soon took more important positions in the field of medical jurisprudence. Among his outstanding publications were such standard books as "Medical Malpractice," published in 1943; "Doctor and Patient and the Law," published in 1949. A revised edition of the latter is now in preparation. In addition to several chapters written for textbooks, more than 150 of Dr. Regan's articles on medico-legal subjects have been published in the various American Journals.

The position that Dr. Regan held in the field of Forensic Medicine is evidenced by his current membership on the American Medical Association's Committee studying medico-legal problems. He served as liaison and committee representative for the Los Angeles County Medical Association, representing that organization in contacts with lay and public health groups and organizations. He was a member of the attending staff of Los Angeles County General Hospital; on the consulting staff of Hollywood Presbyterian Hospital, Los Angeles; Methodist Hospital of Southern California; and Physicians and Surgeons Hospital, Glendale, California. He was Professor and Head of the Department of Legal Medi-

cine, College of Medical Evangelists, Los Angeles; Clinical Professor of Forensic Medicine, University of Southern California. He was a fellow of the American Public Health Association; Past President and a founder member of the American Academy of Forensic Sciences; Past President of the Los Angeles County Medical Association, and, until recently, Legal Advisor of the Los Angeles County Medical Association. During the past fifteen years he lectured before national associations, which include: American Medical Association; American Psychiatric Association; Society of Oral Surgeons; American College of Surgeons; Section on Pathology, American Medical Association; various hospitals' associations and the American Academy of Forensic Sciences; and to State Medical Associations over practically the entire country and to many joint meetings of County Medical and County Bar Associations. He made himself readily available to lecture on Forensic Medicine to a wide variety of groups throughout the nation, often traveling at his own expense.

As the importance of Forensic Medicine became better understood in this country, Dr. Regan was able to impress the universities sufficiently so that additional time was given this subject on curriculums in the medical schools throughout the United States. Throughout this country and abroad, he was respected and admired for his remarkable ability and many contributions in his field.

Forensic Medicine in its present status owes a deep debt of gratitude to Dr. Regan for his ceaseless efforts in the accomplishment of establishing it as a specialty in its own right.

He suffered an acute attack of coronary thrombosis the latter part of November and died December 3, 1955. Medical jurisprudence has lost its best crusader, champion, and most persuasive advocate for the rights and justice of man. His passing has left a void not soon to be filled, and never quite so well.

Dr. Regan leaves his widow, Isabel, a son, Louis J. Regan, Jr., and six grandchildren. With all of his numerous accomplishments, he retained a modesty, simplicity and integrity that endeared him to a host of friends.

Throughout his entire career he was constantly advocating and practicing the good in man. His teachings and writings were the signposts guiding us in the attributes of the good

in law, and the moral and legal understanding of "Right and Wrong."

Not by years but by disposition is wisdom acquired. A wise man is strong; yea, a man of knowledge increaseth strength. Thus in the words of the Sages is reflected the life and work of Dr. Louis J. Regan.

FREDERICK D. NEWBARR, M.D.

Irresistible Impulse and Criminal Responsibility*

Henry A. Davidson, M.D., Cedar Grove, New Jersey

In a dozen states (2), irresistible impulse is a defense to a crime committed because of such impulse. On the face of it, one would expect irresistible impulse to be a defense everywhere. It seems illogical to hold a man accountable for doing what he could not avoid doing. Yet most jurisdictions are skeptical of this defense. Every man is accountable for restraining antisocial impulses. "There could never be a conviction" explained one Judge (1) as far back as 1881, "if the guilty party could assert an impulse produced by some pressure which he could not resist. To restrain such impulses is the duty of all man. He who yields to them, must take the consequences."

If an epileptic, during a convulsion, strikes a bystander, no one charges the epileptic with assault. If an armed bandit compels a victim to remove his pants, no one charges the victim with indecent exposure. Yet when the compulsion arises, not from external duress or physical disease, but rather from inner emotional disturbance, the law—in most states—exacts its pound of flesh.

Should the criteria of criminal responsibility therefore be enlarged to include irresistible impulse as a defense? Is this medically sound, socially desirable, legally workable?

Confusion arises because, from the doctor's viewpoint, there are three different kinds of impulsive behavior: the psychotic, the neurotic, and the normal. Legal precedents generally fail to distinguish between them. So far as I know, this division of irresistible impulse into these three categories

* Presented at the Annual Meeting of the American Academy of Forensic Sciences, February 25–27, 1954, Chicago, Illinois.

has not been previously published (3). The three types of impulse are:

(a) Explosive reactions in psychotic (insane) persons.
(b) Obsessional compulsions in neurotics (example: pyromania).
(c) Rage reactions in persons with no psychosis or psychoneurosis.

Psychotic Impulses

Legally, the irresistible impulse doctrine was designed to apply to the first (psychotic or "insane") category. When legislators and judges speak of irresistible impulse, they mean the psychotic (insane) defendant. But to most doctors, the phrase suggests the compulsions of the compulsive or obsessional neurotic. To the doctor, irresistible impulse usually means psychoneurosis; to the lawyer, it usually means psychosis. The two professions are not speaking the same language here.

Many court decisions sustain the position that irresistible impulse was intended to limit its application to psychotic (insane) defendants. One of the first relevant decisions in Anglo-American law (1868) included the phrase:

> "If it should be definitely established that the defendant had an unsound condition of the mind, a *diseased mind*, in which he is, by *insane* impulse proceeding from the *diseased intellect*, irresistibly driven to commit the act, then the law must modify its ancient doctrine." (4)

The italicized phrases (italics mine) make it clear that the court meant insanity, and nothing short of it. Even when the accepted medical term for a neurosis is used, the legal decision often makes it clear that a psychosis is contemplated. "Kleptomania is a species of insanity." (5) That's the way the court put it, obviously confusing psychoneurosis (kleptomania is a neurosis) with a psychosis (only psychoses are species of insanity). Another court used this wording: "He was, in consequence of his *insanity* wrought up to a frenzy which rendered him unable to control his acts." (6) In another case, the defense was acceptable because, in the court's words, "there was such overwhelming violence of *mental disease* that he was not a responsible moral agent." (7) The Parsons case is generally hailed as the one which liberal-

ized the ancient and rigid doctrine. But even here, the court admitted only such impulse as was rooted in psychosis, for the first criterion was: "Was the defendant afflicted with *disease of the mind,* idiotic, or otherwise *insane?*" (8) Only if the answer were "yes" did the other part of the rule (the exculpation by reason of irresistible impulse) operate. The test was, "whether by reason of this *mental disease* he had lost the power to choose between right and wrong." In another state (9) it was held that the "inability to refrain from the wrong" must be due to "impairment of the mind by disease." At the other end of the country, a court (10) held that irresistible impulse was an acceptable defense only when it rose from *disease.* In another jurisdiction, (11) the exculpatory phrase was "loss of power to resist temptation" but this was irrevocably tied to "insanity." The loss of self-control was held exculpatory in another state (12) only when it was "due to the overwhelming violence of mental disease." The earliest Pennsylvania case on point (13) requires that "the *insanity* be so great as to take from the subject his freedom of action." And many other similar decisions can be cited. It will be noted that throughout these there is one common thread: the irresistible impulse is part of "mental disease" (which means insanity), and not part of psychoneurosis. A handful of decisions can be found which seem to indicate that in an occasional jurisdiction, irresistible impulse by itself (not as a symptom of insanity) is exculpatory. But usually there is here an implication that the court simply assumed, as a matter of course, that the psychosis (insanity) has been established.

It seems clear that when the doctrine was first laid down, the courts meant that irresistible impulse, if it excused at all, would be such an impulse as came out of a diseased (psychotic, insane) mind.

Mens Rea and Psychotic Impulse

Probably no one wants to hold accountable an insane person who commits an unlawful act under the compelling imperative of his insanity. Do we need a special irresistible impulse doctrine to protect such a person? Or is he adequately protected by the general law, including the McNaghten rule itself?

It seems to me that a person with a definite psychosis (actual insanity) who does an act because of that insanity, and does it specifically because of a compulsion rooted in the psychosis, is not acting voluntarily. *Mens rea* means guilty mind, or intent to commit the act. If there is no *mens rea,* there is no crime. Obvious examples would be injuries caused by movements during convulsions, sleepwalking or choreiform twitches. In these examples there is no intent to harm, no *mens rea,* no crime. *Mens rea* implies a free and deliberate choice to do wrong: this surely does not describe the compulsive act of a psychotic. Free agency is destroyed by the fabric of a psychosis. A sane defendant would be acquitted if he performed an otherwise unlawful act because of physical infirmity, convulsion, severe duress, hypnotism, sleepwalking or mechanical obstacle. If a sane defendant is thus excused, presumably and *a fortiori* a psychotic person would not be responsible. On these grounds alone, it seems to me that even in the absence of a formal irresistible impulse doctrine, a psychotic defendant is not held accountable by sophisticated courts when the act was the result of a psychotic delusion.

There is another reason that a psychotic impulse could lead to an acquittal—though I think this is less clear cut than the lack of *mens rea.* That is that the existence of a psychotic compulsion tends to obliterate any meaningful sense of wrong-doing, hence should be exculpatory even under the classical "right and wrong" test.

In some states where, in theory, irresistible impulse is *not* a defense, courts have admitted the defense in practice. What the courts have done is to have found that the psychotic impulse overrode judgment, reason, cognition and appreciation of the sense of wrong. Glueck (14) cites cases from California, Kansas, Mississippi, Iowa, Texas and West Virginia which reflect this. Presumably in these states, a defense of irresistible impulse would be rejected if the impulse were of "neurotic" intensity. In discussing this with me, a southwestern Judge said: "A mere irresistible impulse is no defense in my state. But an impulse made irresistible on account of insane frenzy is a good defense." The phrase "mere irresistible impulse" shows a serious underestimate of the potency of neurotic compulsions. But apparently it is sound law.

Sir Fitzjames Stephen (15) as far back as 1883 argued that the McNaghten rule was broad enough to protect a psychotically-rooted impulse. Thus, he said:

> "The man who cannot control himself is in that state because he is guided by motives which immediately press upon his attention. Thus, the power of self control means a power to attend to distant motives and general principles and connect them rationally with the act under consideration. A disease which so weakens the sufferer's powers as to prevent him from attending or referring to such consideration, or from connecting the general theory with the particular fact, deprives him of the power of self control. How does any one know that an act is wrong except by comparing it with general rules of conduct which forbid it? If he is unable to apply such rules, how is he to know that what he proposes is wrong? *The absence of the power of self control involves an incapacity of knowing right from wrong.* Knowledge and power are the constituent elements of all voluntary action. *A man who cannot control himself does not know the nature of his acts.* A man who does not know the nature of his acts is incapable of self control." (Italics mine).

On this reasoning, as well as through the *mens rea* doctrine, one is led to the conclusion that a psychotic defendant is not, by the traditional McNaghten rule and by the general principles of criminal law, responsible for an act which results from a psychotic (insane) irresistible impulse; and that no specific doctrine is needed to spell out such a defense.

Neurotic Impulse

Psychoneurosis, not psychosis, is the real battleground over irresistible impulse. Pyromania, for example, in certain sex delinquencies, in neurotically motivated writing of poison pen or obscene letters, in behavior like this, there is, by definition, no insanity. To the juror, often to the judge, an "irresponsibility" defense is misinterpreted as an "insanity" defense. (Actually, irresponsibility not insanity is the defense.) If the defense counsel wants his client found "irresponsible," he tries to get the judge and jury to think of his emotional state as some species of insanity. An obsessional neurotic is *not* insane. But the defense, realizing that it is easier to get an

acquittal if the "psychosis" label can be attached, strives mightily to emphasize the abnormality of the defendant's emotional state, hinting that it is somehow akin to insanity. A neurotic in the grip of an irresistible impulse is obviously not in a normal or healthy state of mind, and the defense effort is to equate an abnormal or unhealthy mind with an insane one.

With respect to neurotic (that is, sane) impulses, there are three questions to be answered: (1) do these impulses really exist? (2) might they lead to violent or criminal behavior? and (3) what should be the medico-legal status of a neurotic defendant in terms of accountability to the law?

I do not suppose that any educated person denies the existence of neurotic obsessions and compulsions. A patient might have a powerful, an overwhelming, urge to do something absurd—an urge so overriding that it seizes consciousness while it dethrones conscience. It may well be that the penalty for abstaining is a severe one: nauseating sickness, choking panic or almost-unbearable anxiety. All this is true.

But is such a compulsion irresistible? In any 100 per cent sense, no. If the patient will pay the price in terms of his anxiety, he can resist—and in the presence of others, he often does hold his hand back. Assume an obsessive-compulsive about to set a fire. A bystander walks by. The patient abstains. If the bypasser remains, the patient waits. His anxiety may mount, he may get palpitations, he may feel faint, he may well-nigh drown in his anxiety. But he can and does wait. The impulse is resisted: It is, therefore, not irresistible. This is the basis for ancient but useful Bramwell formula: would the defendant have committed the crime had there been a policeman at his elbow? With some psychotics—indeed, with McNaghten himself—the answer is "yes." So compelling and irrational was the urge that the presence of the policeman would not stop him. When a paranoic is being apprehended, he often assaults the policeman who has come to take him. Here the impulse is not resisted in the presence of a policeman. With neurotic compulsions, however, the answer to the Bramwell query is: "no." The defendant would not have committed the crime had there been a policeman at his elbow.

So, with neurotic impulses, the answer to the first question is that such an impulse is not absolutely irresistible, only

relatively irresistible. The patient can abstain at the cost of considerable anxiety. One must avoid sanctimonious moral judgments here. Only he who has suffered such panic, such miserable anxiety, has the right to say smugly: "if you had the will power, you could have refrained. Since you yielded to the impulse, you must suffer the consequences."

The second question is whether neurotic obsessions can lead to antisocial behavior. Wertham (16) says "no." Here is the way he puts it:

> ". . . There is, with one exception, no symptom in the whole field of psychopathology that would correspond to a really ungovernable or uncontrollable impulse. That exception is an obsessive-compulsive neurosis. . . . Compulsions play no role in criminal acts. It can be stated flatly that compulsions are always unimportant and harmless acts. A patient may have to count the windows, to wipe off the doorknob, to avoid stepping on cracks in the pavement. But he never has to commit a truly impulsive criminal act. . . . *In the whole literature of psychiatry, there is not a single case where a violent act constituted a symptom in an obsessive-compulsive neurosis.* The 'irresistible impulse' rule is socially backward. . ." (Italics mine).

This dogma sounds so sweeping that one's immediate reaction is that it simply cannot be true. Pyromania is a well-known compulsion. Most psychiatrists would say it was an obsessional or neurotic kind of compulsion. Arson is a violent act in any lexicon. Yet here Dr. Wertham insists that "there is not a single case where a violent act constituted a symptom of an obsessive-compulsive neurosis."

The difficulty here is this: there are actually two kinds of neurotic impulses, one being a ritual to protect against guilt and one being a sadistic act to bring on self-punishment. In the first category, the compulsive act must be harmless. Its whole purpose is transfer of an affect from guilt-laden to innocent behavior. This innocence is of the very fabric of the neurosis. In Alexander's happy phrase, (17) the compulsion is "a bribe to the superego." The patient can say to his conscience: "I am just counting cracks or washing my hands; an innocent ritual; really I have not sinned; see how innocent this act is, and how busy I keep myself with it."

But these compulsions never reach the court; within this framework, Wertham was right.

Unhappily, there is a whole calendar of crime which can be reasonably attributed to neurotic compulsiveness. Kleptomania and pyromania, to mention two, are antisocial and dangerous. Other neurotically rooted impulsive violations include open lewdness, writing of poison pen letters, sending obscene letters through the mails, indecent self-exposure, sexual acts against children, voyeurism and the like. These are antisocial and some are very traumatizing to their victims. So the answer to the second question must be: yes, neurotic compulsion can lead to antisocial behavior.

The third question concerns the accountability of these patients. This is a matter of social policy rather than medical definition. However, certain aspects of it are worth a little reflection by the doctor. I think here of the moral, the practical, the psychological and the social aspects of the impulses of the neurotic.

Neurotic Impulsiveness: Moral Aspects

Generally speaking, a man is accountable for doing what he knows he should not do. The psychotic has, as a rule, such distortion in reasoning, that he does not, in any meaningful sense, appreciate what he ought and ought not do. His premises may be wrong, his values may be wrong, his method of reasoning may be wrong. But here, our concern is with the neurotic. In the neurotic, reason is basically normal. Now the law is more interested in intent than in motivation, whereas the psychiatrist is more interested in motivation than in intent. Thus, in a shoplifting, the law wants to know whether the arm moved towards the counter with an intent to lift; and if so, whether there was intent to avoid payment. These intents are legally necessary to establish the crime; and indeed, completely establish it. If there was no intent, there would be no crime. The psychiatrist, however, is little interested in the immediate intent. He wants to know the meaning-value, in a deeper sense, which the stolen object had. Kleptomania is a symbol—a symbol of what? If the shoplifter does not use what he steals, what then is his basic motive? These are the psychiatrist's questions.

A person in the grip of a neurotic impulse does *intend* the crime. He intends to steal, or to expose himself or to set a

fire. Man is morally accountable for what he intends to do and for the harm which the intentional act caused others. The neurotic compulsive meets those criteria and would seem to be morally responsible for the effects of what he intends to do. This is, I confess, a self-righteous view. But moral judgments are always righteous. Judge Coleman (18) was voicing the righteous judgment of the public when he said, in this connection: "To restrain antisocial impulses is the duty of all men. He who yields to these impulses must take the consequences."

It is, by this reasoning, no answer to say that the neurotic must act because otherwise he goes into a panic. From the viewpoint of one who has never known an obsessive compulsion, the ill-effect of restraint is simply a discomfort, like not smoking in church. If abstinence from smoking all Sunday morning makes you tense or anxious, you are expected to put up with that discomfort, rather than try to sneak a smoke by keeping your head and mouth down in the pew. The neurotic says he feels sick if he does not set fires. The righteous answer is that if I refrain from stealing, I may have to go hungry; if I refrain from rape, I have a lot of unsatisfied tension. This does not excuse theft or rape or smoking in church, so it should not excuse you for setting fires. Better hold back your hand and have the anxiety than yield to the tension and burn up a child.

This assumes that a neurotic compulsion differs only in degree from normal temptations. Let us suppose that Mr. A. gets sexual satisfaction only out of exposing his genitalia to unwilling little female observers. Mr. B. gets satisfaction only out of homosexual contacts. Mr. C. gets satisfaction through hetero-sexual contacts using the stereotyped channels and techniques. Now Mr. C. is expected, no matter how vigorous his libido, to curb his sex impulses until he finds an adult female willing to copulate with him in a private place. Mr. B. is expected to find a willing adult partner with whom to share his sodomy in private. If Mr. B. seduces a child, forces himself on an unwilling adult or engages in sodomy on the city hall steps, he is held accountable. So, Mr. A. is expected to find a willing adult partner and a private arena for his exposure of his genitals. All this assumes that Mercier is right in saying that "perverted desire is no more urgent than normal desire. The sexual pervert is as responsi-

ble for an act which gratifies his desire as is the ordinary fornicator or adulterer." (19)

If we were to exculpate the homosexual or the self-exposer because of the over-riding strength of his compulsion, we would simply be offering a premium to the person who does not control his desires and laying a penalty on the ordinary citizen who does. Society expects the same restraint of a perverted drive that it does of a normal compulsive urge like hunger, sexual desire or thirst for water.

Finally, the moral issue considers the victim too. Thus it has been argued that this country was wicked to punish the war criminals in Germany and Japan. These men were, the thesis runs, driven to their atrocities by factors deep within themselves. Society made them sadists. Therefore we must forgive them for Society is the guilty party. The victims of the gas ovens and those who suffered their other unspeakable brutalities may not appreciate this high-minded charity of judgment. Still it is a laudable effort to be objective. To identify oneself with this, picture that your daughter or favorite niece was ruthlessly raped and then her belly torn open by a neurotic assailant. To view this objectively, you should try to protect the assailant from prison and agree that the proper disposition would be enrollment in a mental hygiene clinic.

Society itself feels tense, disturbed and restless when a brutally aggressive defendant is disposed of in a gentle fashion. Such disposition breeds a sense of outraged righteousness: an ignoble reaction, perhaps, but a realistic part of the moral judgment of our people. The moral answer is that the neurotic is expected to curb an impulse to hurt others even if he has to hurt himself in the process.

Neurotic Impulsiveness: Practical Aspects

Would a law which acquits the compulsive neurotic actually increase the crime rate? Conversely, does a law which punishes him have any effect in deterring others? There is no evidence that there is any more crime in the dozen states (2) which recognize irresistible impulse than in the three dozen which do not. In this rather naive sense, the doctrine is not producing a crime wave in states where it is in effect. But this is a crude method of proof. Judges are not concerned

with this as a doctrine. For example, the law may exculpate crimes resulting from irresistible impulse. A jury may ignore that defense and bring in a verdict of guilty if they, the jury, think that defendant ought to be held accountable. There is little here that can be appealed. The defense would appeal on the grounds that they had proved irresistible impulse and that the jury had no right to ignore that proof. The appellate court is likely to say that whether such an impulse existed is strictly a jury question. In finding the defendant guilty, the jury indicated disbelief in the existence of that impulse in this defendant. This kind of factual finding is rarely disturbed by a higher court.

The existence of the doctrine does make it easier to spare certain defendants from being indicted. It makes it easier to have an indictment quashed. I doubt if it has much practical effect on juries.

Conversely, in a state which denies the defense, a jury could find an accused "not guilty" if they thought he was helplessly in the grip of an irresistible impulse. This verdict, though legally unsound, could not be appealed. It would never enter the law books and the state's official position on irresistible impulse and it would remain unchanged even though the jury had, in practice, changed it.

To adopt such a doctrine might have some slight effect in that, each year, one or two defendants convicted by jury might be freed on appeal. But the number would be too few to affect the crime statistics.

Neurotic Impulsiveness: The Psychological Issue

There are two kinds of neurotic impulses. One type of compulsion is an innocent ritual developed to dam back aggressive or sinful tendencies. The other compulsion is itself an aggressive, sadistic act rather than the denial of one.

Ask a psychiatrist what explains a neurotic compulsion and he will usually tell you that it is a sort of harmless ritual intended to relieve the pressure to commit a sin. But when the lawyer or criminologist mentions irresistible impulse he thinks of something like a pyromania or a kleptomania and not of an innocent whim like counting windows. The lawyer, thinking of these aggressive impulses, asks the psychiatrist: "How do you people explain neurotic compulsions and what

makes a person repetitively perpetrate an act he knows is foolish?" The expert gives the right answer: it's a ritual to protect him from guilt feelings. The trouble is that the doctor is thinking of a compulsion to wash hands. The lawyer is thinking of arson or shoplifting.

The type of impulse which gets into court is not hand-washing or window-counting. It is an overtly aggressive impulse. It would be absurd to say that such an act is to protect the patient from doing something sinful. The act itself is sinful. There is panic if the act is not performed: there is delayed guilt and recrimination if it *is* performed.

The patient acts as he does because of an unconscious wish to be punished; or because he has regressed to an anal-sadistic level and the act is an expression of a sadistic drive. This is the neurotic compulsion which gets into the criminal courts.

Several observers have commented on the fact that the "I" who does the compulsive act is not the "I" on trial. Wechsler (20) for example, speaks of the compulsion as a "foreign body which does not belong to the ego." Mercier (21) makes a similar analogy. He refers to a patient with chorea who stuck his fork into his own neck instead of into his mouth because he could not control the choreiform twitch. He speaks of a defendant who compulsively uttered obscene words. "My tongue and my mouth utter the words; but 'I' did not utter them for I had no intention of uttering them. I had no more part in the act than had the choreic in sticking the fork into his neck."

This is the concept of the nonparticipation of the ego in a compulsive act. The courts don't want to sentence the wrong person. If the "I" which did the act is not the "I" which is being tried, then it looks like a miscarriage of justice to find him guilty. The analogy to the innocent chorea victim is attractive but faulty. The chorea patient did not intend to stick the fork into his neck. The other defendant *did* intend to utter the words. To be sure, he did not *want* to say the words: but responsibility inheres in intention and not in motive.

The analogy breaks down in a practical sense too. Consider a man who has made himself drunk in order to get the uninhibited rashness needed to commit an assault. He is so drunk that he develops an amnesia, either that of hysteria or that of pathological intoxication. On trial, he has not the

slightest recollection of committing the crime. He will never, so long as he lives, recall it. It is as if you yourself were told that last night you had broken into City Hall and stolen the mayor's gavel. You know perfectly well that you did no such thing. No one can make you ever believe you did. Yet you are convicted. So, it might be argued, that the man on trial is not the man who committed the crime. This doctrine would not be accepted by the public or by the courts.

Neurotic Impulsiveness: Social Aspects

The social aspect of this problem is one which seems to be of little concern to either legal or medical specialists. Assume a jurisdiction where a neurotic impulse is a defense. Here is a person who feels compelled to set fires. The psychiatrist explains that this man sees fire as a symbol for love and passion. When his sexual tensions mount, this is his only effective outlet: to start and watch a fire. If he is prevented from lighting the fire he gets sick and goes into almost unbearable panic. Legally, his neurotic compulsiveness is, we have postulated, a complete defense. Medically he ought to be acquitted because he could not help doing what he did. Now suppose you protest that to set this man free would be to set a firebug loose on a sleeping city, to invite possible destruction of your home, to condemn to the horror of being burned alive some innocent child. This is no over-dramatization. Turn a pyromaniac loose tonight and it could happen. The judge would have to say "I can't help turning him loose. The law says the man is irresponsible, so he cannot go to jail. The doctors say he is sane: therefore he cannot be committed to a mental hospital. Hence, I have to turn him loose." This refers only to jurisdictions where irresistible impulse is a defense.

The doctor would have to agree to this. Surely the defendant is a sick man, he would say. It is wrong to send a man to jail just because he is sick. Jail is not the place for psychotherapy. Suppose you answer: "But, doctor, if the man is turned loose, a serious social problem is produced." The doctor could well answer that it is his sworn duty to protect and treat the individual patient, and that society has its own officers and protectors.

If neurotic compulsiveness is a sound defense, legally, then no one seems to take responsibility for the social problem

produced. The legislature *could* take that responsibility by enacting that a neurotic compulsion would not be a defense, and thus directing that the neurotic pyromaniac or kleptomaniac go to jail. Or the judge could take that responsibility by sentencing such a defendant to jail, if, in spite of the law and the plea, the jury convicted him. But then the legislator or the court would be branded as old-fashioned, punitive and driven into severity because of some unconscious guilt in the judge or legislator.

It seems clear that if the neurotic compulsion is traumatizing or dangerous, the defendant should *not* be exculpated. From the patient-oriented doctor's viewpoint this may be a harsh gospel. But the protection of society really does require it. The only alternative would be legislation permitting commitment to a hospital of a nonpsychotic psychasthenic who had violent antisocial compulsions. And this, I think is a more dangerous expedient than the other. The compulsive neurotic with a socially dangerous impulse will not voluntarily lock himself away from society. This has to be done by compulsion until he is cured, and the compulsion either has to be a legal one or a medical one; that is, a hospital commitment.

In this connection, I think of a footnote in Glueck's book (22): "Baron Alderson" he writes, "anticipated the modern doctrine. A man might say that he picked a pocket from an uncontrollable impulse. In that case, the law would have an uncontrollable impulse to punish him."

At first, this sounds like a gag which a speaker can pull to show how smart he is or to get a laugh. But the concept is a serious one. Shorn of its humorous overtone it means this: an offender commits a crime because he cannot help himself, because of some imperative in him that compels him to act that way. But noncriminals also have imperatives that cause them to act in certain ways. Legislators enact punitive laws, juries find defendants guilty, and judges impose sentences because all these people also have emotional drives. If, under certain circumstances, it is human nature to commit murder, then it is also human nature to feel frustrated when another gets away with murder. The mental hygiene of society may require punitive action. Every unpunished assault makes the streets less safe for you and me. Every unrequited murder makes every man's life a little less safe.

Much has been written about the unconscious determinants of criminal behavior. Little is written about the emotional need to see crime punished. One of the few who has attempted to tackle this head on, is Abrahamson (23). Conceding that punitive legislation subserves emotional needs in the public, Abrahamson writes:

> "It is irrational for the offender to behave as he does. It is also irrational for us to take a condemning and destructive view of him. When he is wrong, it does not help matters for us to be wrong too. . . . If every one carefully thought over his own feelings towards the delinquent, he might then understand his own punishing attitude. . ."

Dr. Abrahamson also concedes (24) that "if there were no punishment, crime would be so frequent that there would be no end to it." This is bewildering. On one hand, we are told that without punishment, there would be no end of crime. On the other, we are told that crime is the result of deep-seated emotional forces and that thus the delinquent is really sick and should not be punished. This trail of thought leads to a dead end.

Rage Reactions in Otherwise Normal People

The third type of irresistible impulse is the rage reaction in the otherwise normal person. (The other two were psychotic and neurotic impulses.) These rage reactions include sudden fits of temper, the fury of the cornered psychopath, impulsive violence in people who are, clinically, neither neurotic nor psychotic, and the violence of alcoholism. In varying degrees, all people are subject to attacks of rage, potential or expressed, which lead to the thought that some sharp, severe, brief bit of violence would be a great relief. So a man might look through a store window and see something he wants with such longing that, for a moment, he is tempted to break the window. A man with a strong sex drive might be in a subway car at night alone with a strange and exciting girl; and to him comes a strong urge to copulate with her by force if necessary. Driving an automobile there may be the urge to step on the gas and go 100 miles an hour when the road is clear and the engine is purring smoothly. There may also be the urge to choke the bridge partner who

has just trumped your ace, to knock down the man who has said something sarcastic.

But somehow most of us restrain the antisocial impulse. Society says that punishment will be visited on the man who does not restrain it. If the phrase "irresistible impulse" be defined with enough generosity, all of these rage and alcohol reactions, would fall within the definition. Since I have already indicated that the irresistible impulse doctrine should not apply to psychoneurotic compulsions, I need only add that *a fortiori,* it should not apply to the rage reaction in the otherwise normal person.

There is little to be said about brainstorms, about the allegedly temporary insanity, that comes in a trice and leaves without a trace. The psychiatrist can recognize a psychoneurosis, for psychoneurosis is a way of life, not a transient reaction. He can recognize a psychosis, for that is his simplest professional duty. In the absence of psychosis or neurosis, the brainstorm which leads to violence may be due to alcohol or to frustrations. It is playing with words to call it "insanity."

Summary

Irresistible impulse is a defense in a dozen states (2). Many believe it should be a defense in all. As contemplated by the law, the phrase "irresistible impulse" refers, or was originally intended to refer, only to the compulsiveness of an active psychosis. This is what the law means when it throws the protecting cloak of this doctrine around a defendant: it means he was insane and his impulse was rooted in that insanity.

The doctor, however, sees two other levels of such impulses. In addition to the psychotic impulse, he recognizes a variety of neurotic impulses, and also rage reactions which lead to violence in otherwise normal people.

It is generally agreed that a person who commits an antisocial act because of a psychotic impulse should not be treated as guilty. No special legal doctrine is needed to protect him. The only standard of responsibility in most states is the McNaghten rule. This does not specifically exculpate impulsive behavior. But it does shield the person who does not appreciate the wrongfulness of his acts. A psychotic in the grip of a frenzied impulse cannot weigh the legal or moral

rightness of his acts. He is thus protected by the general rule. Furthermore, his act is not voluntary because the psychosis destroyed his free agency. An involuntary act never amounts to a crime. A special irresistible impulse doctrine is not needed to protect the psychotic defendant.

There seems to be no justification for exculpating the otherwise normal person who commits a crime because of a rage reaction, acute alcoholism or the frenzied fury of a cornered psychopath. If such acts were not considered crimes, then no one would ever be responsible for anything. All antisocial acts are the result of emotional drives which—by the definition of "antisocial"—are also, in a sense, abnormal drives.

The neurotic compulsion is at the nub of the problem. At this level there are two very different kinds of neurotic impulsiveness. Failure to recognize this has caused much confusion. Traditionally, the psychiatrist thinks of an obsessive-compulsive act as an innocent ritual needed to protect against the guilt feeling generated by the patient's thought of sin. All such obsessions must be harmless since their purpose is to display innocence. The phrase "neurotic compulsiveness" or "neurotic irresistible impulse" in the psychiatrist's mind conjures up the image of one of these harmless rituals.

But to the lawyer or judge, it suggests pyromania, kleptomania, an urge to send obscene or poison-pen letters, a compulsion towards self exposure or some other traumatizing act. So the lawyer and doctor are not talking the same language when they speak of neurotic irresistible impulse. The doctor thinks of the harmless obsession, the lawyer thinks of the dangerous impulse.

It would be socially hazardous to exempt the latter group from accountability. Under these circumstances, offenders cannot be committed to hospitals because, by definition, they are sane.

If they are considered legally irresponsible, they cannot be detained in jails or prisons either.

If a person has a powerful, repetitive neurotic urge to do violence, the protection of society requires that he be restrained. Unless he is held responsible, he simply cannot be lawfully restrained.

It is technically incorrect to say that such a person is in the helpless grip of an irresistible impulse. The neurotic with

this type of impulse can, and usually does, control it in the presence of a third party.

In controlling the impulse, the neurotic usually hurts himself. In yielding to it, he hurts others.

An impulse which can be resisted in the presence of a third party, is not irresistible.

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Some Medico-legal Problems Connected with Electric Shock Treatment*

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Introduction

It is quite possible that many medico-legal texts will become obsolete in the very near future. The work, for example, of Selye on stress (1) plus the newer medical and operational philosophies (cybernetics), as well as findings in cases which have been given electroshock treatment, will alter the whole picture of Forensic Psychiatry. For that matter, if the new teachings of medical and psychologic science be followed, there must be a new code of diagnosis and treatment which would cut the time of judicial hearing, make the apprehension, trial, and treatment of the offender more rational and accurate than ever before, and vastly cut the cost in time and money of our present criminal procedures to a new minimum.

The problems which arise from the use of electroshock point out why it will be necessary to have a new code of judicial procedure for these cases, if not in others. There are certain features resulting from electroshock treatment which are bound to change the picture which the law sees of the alleged offender.

Electroshock has several aspects which demand changes in our legal methods. First of all, cases, to get maximum benefit, will have to be brought to the psychiatrist for treatment

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before they can even have a preliminary hearing. Cases of depression, and, to some extent, compulsion neuroses with homicidal tendencies, should be put under immediate treatment and must not wait for weeks for judicial determination for hospitalization. Whether a person has committed a crime or not doesn't change his medical nature which needs treatment whether the case be brought up in the County Court for commitment or whether it be brought up in Criminal Court for commitment to a hospital for the criminally insane. The outlook remains the same in either case and should eliminate the need for trial and rehearing anywhere from months to years after the crime was committed when the person gets well. By that time, at least under our current procedure, it is very difficult to collect the witnesses.

The question is whether there is definite evidence that the offender is sick and needs treatment, whether he is a criminal or a noncriminal. The determination of what is to be done with him in the future must be predicated upon what we know about the future behavior of all individuals in his diagnostic group who have been treated. If he continues to be a risk in the community, he must be segregated for years even if he is a misdemeanant, and if he is not a risk, nothing is to be gained by hospitalizing (imprisoning) him after his treatment no matter how serious his offense.

The Need for Immediate Treatment

Although suicides and suicide attempts are carefully concealed from the general public, so far as that can be done, a few stories about how offenders have destroyed themselves, or attempted to do so, do get into the news. Since most of these are not persons who carry news value, the stories about them appear as squibs or do not appear at all, but there are still large numbers of people who have been arrested for offenses, attempted offenses, or even threats, who, being in a depression, do attempt suicide. These people should be immediately put into prison wards or similar places to be given proper medical treatment.

The idea of immediately treating the mental case has not been accepted until very recently (2). However, a number of years ago, in Detroit, and fairly recently in Winston-Salem, North Carolina, we had two cases where legal procedures

were held in abeyance while the patients, who had each murdered her own child, were immediately put under treatment. One of these cases was diagnosed as a depression and after four weeks of shock treatment recovered, and the other case was diagnosed as schizophrenia, but to my mind, and I know many of the details of the case which did not appear in the press, this was also a case of depression.

Since, under the law, these women were treated and were then to be brought back for trial, a number of medical-legal questions come up. If they were psychotically depressed when they were examined by the psychiatrist to such an extent that they required shock treatment, why bother to try them? Even though this be homicide, why not dismiss the charges? In the first case, the judge and the prosecuting attorney were willing to let the case drop but the Michigan law at that time required that all persons who were accused of homicide, even if acquitted, be sent to the State Hospital for the criminally insane to remain indefinitely for, under this law, there is no provision for even gubernatorial clemency.

In the second case, the prosecutor was unwilling to drop the charge even though it was quite obvious that under ordinary tests of insanity, this patient was not rational at the time the crime was committed. I do not know, as yet, whether the patient has recovered sufficiently to be brought to trial, and if so, what happened.

This immediate institution of treatment is something entirely new in the courts. Most sanity rulings have grown up through ponderous additions and subtractions to the common law and through the rulings of McNaghten's case.

Since it made no difference to the patient, prior to the era of physiological treatment of the psychosis, whether a long or a short time be taken before he was sent to the institution for the criminally insane, court procedure was permitted to take its time. Patients were not arraigned for a year, and sometimes even longer, in some jurisdictions. The rushing of the patient to a psychiatrist to prevent suicide and to cure his depression, sometimes within twenty-four hours of his arrest, is something entirely new to jurisprudence. Even commitment cases in most jurisdictions take a week, being held without treatment pending a sanity hearing. I do not think a delay of a week or so in most cases will make it neces-

sary to give more than the usual number of treatments, but certainly the law's delay of a year or even a few months is something that cannot be tolerated when the issue is one of restoring a healthy-minded person to the community in reasonably good condition instead of having a chronic mental invalid on the hands of the community for support and maintenance for many years. Judicial delay can no longer be tolerated.

I am in complete accord with those who believe that rushing an offender off to prison without due regard to his civil rights is wrong. However, careful observation and diagnosis of a mentally sick person does not require, in most cases, all of the investigation and study that a criminal trial does. I think it is reasonably safe to say that even if the person didn't need psychologic treatment, no harm would be done by giving it to him or to her. Certainly the clinical picture is such that an experienced—certainly certified—psychiatrist should know what cases require the treatment. Often if the patient does not seem to require it immediately, he will show such signs of depression to even a jailer—with a certain amount of humanitarianism in him—who will be able to report the patient's condition.

The Patient as a Witness; the "Amnesia"

While it is certainly possible to give patients shock treatment without producing a serious amnesia, the psychotic patients who commit a crime are usually sufficiently serious so that a great deal of shock treatment is necessary. While certain types of apparatus, particularly those made by Reuben Reiter, seem to give less amnesia than others, there are psychiatrists who prefer the non-Reiter kind of electroshock treatment because of the belief that fewer treatments are required and the results are more permanent than with other types of equipment. These beliefs are open to question and it seems reasonable to believe that techniques will be developed which do not result in too much amnesia.

I feel, in post-electroshock amnesia, we have a number of medical and legal matters to look into. First, how much reliance can we place in the testimony of a witness who has had shock treatment between the time the event occurred and the time that he testified?

Again, we have the matter of the law's delay. If a person is going to need shock treatment in the near future after the event that he has observed, we must ask ourselves how valid his testimony would have been in the first place. I have oftentimes wondered why attorneys at a hearing did not routinely ask people, especially witnesses and talesmen, whether they had had shock treatment within six months of the time that the case was being brought to court. Even small lacunae of memory defects can be present in the mind of the witness and there may be a tendency to forget for as much as two or three months after the treatment was given. Many of these small lacunae are almost undetectable but they most certainly have a potential effect upon either the witness's testimony or the decision that the juryman will come to. Perhaps he will have trouble remembering testimony or his mental balance will be such as to carry on prejudices because he cannot remember certain things that were nocuous. It is insufficient, I believe, for the attorney examining the talesman to see if he is to serve on a jury, to ask the usual question, "Have you ever been in a mental hospital?" Oftentimes, some of the most serious psychiatric cases, particularly those in active people who are anxious to keep the community from knowing that they have been mentally run-down in any way, have been given shock treatment in the psychiatrist's office or have been treated by him as an outpatient of a general hospital.

How about the cases who have been arrested, then given treatment like the ones mentioned in the first section of this article? They, of course, are likely to have some very great amnesia. Perhaps they have no recollection of the crime and their defense of amnesia is a perfectly legitimate one, occurring in almost all cases of straight depression or of depressed elements in a psychoneurosis or of the depression seen in some other psychoses such as arteriosclerosis or schizophrenia. These people because of mental disease cannot give any information regarding what happened at the time of committing the crime. I believe it is not proper to make them stand trial. If they are compelled to stand trial as in the cases of the homicidal mothers, the fact that they deny any memory of the offense is likely to prejudice the jury against them. I heard a judge in a similar case harangue a witness saying that she must try to remember an offense which was quite

obvious she had expunged for psychoneurotic reasons and no matter how hard she tried, even under sodium amytal, she could not bring back the memories.

Some Secondary Medico-legal Problems

There are a number of medico-legal issues which are being brought up by shock treatment which, so far as I know, have never been resolved. I know of little that has been recorded in the legal literature. Most of these cases have been settled or have been disposed of in lower courts. The question comes up, suppose the patient is psychotic and is not willing to have shock treatment? What rights does he have? Unless he is declared legally insane, his next of kin has no right to give permission for the treatments, yet, without some kind of permission, what hospital is going to take the chance of treating such a person? What defense does the hospital have if the person refused to sign a permit and has a permit from the next of kin? I think the problem of treating a shock case in the office is a different one. If such a case refuses to come into the office for treatment, and is forced to do so, the physician is perhaps vulnerable. However, in most cases that we treat in the office, the patient comes in voluntarily and he is given plenty of time to await the treatment. He sits around the office and he could walk out or make a protest if he didn't want the treatment. However, the question again comes up about the amnesia. I assume that in many cases, and for some time to come, we shall have amnesia and we cannot gainsay this problem, but if a person is brought in by someone else, no matter how willing he is to have the treatment, how can he be forced to pay for it? This is particularly true if he cannot remember having promised to pay for it at the time that he was given treatment. For that reason, I think that a signed permit and perhaps even a note regarding payment—an agreement which he can see that he made—would be necessary.

Another question comes up regarding recurrent attacks of depression or psychosis which requires additional treatment. This is perhaps more a problem for public administration than it is for the courts but, suppose one has had a previous attack and has been treated by shock treatment. Can a judge or any other public official do good work in the field of law if

he is likely to have a partial or even slight amnesia by virtue of his having a prophylactic shock treatment once a month? Such cases are infrequent but I am now carrying half a dozen patients whose work does not seem to be hampered by the treatment but I think that if I had patients requiring therapeutic shock treatment who worked in the field of law, particularly judges, commissioners, clerks of the court, or those having similar responsible jobs, I would raise the question whether such treatment would invalidate their appointments. However, I am quite properly set against the idea that people should lose their jobs in government or elsewhere because they have had a mental ailment. This doesn't seem any more necessary to me than in the case of throat infection or tonsillitis. However, where the mental strain is as great as it is in the case of a judge, that there may be a recurrence and the possibility of amnesia is another matter. This might very well invalidate a decision in cases coming into the purview of such government officials.

Concerning the problems of examination and licensing, it is quite obvious that with the mental changes which temporarily occur from electroshock treatment, it will be well to consider the application of the laws which pertain to an examination and the licensing of individuals for various types of public employment and vehicle operation.

In the case of motor vehicle operators who have had shock treatment, even after patients are cleared up from the psychiatric and mnemonic standpoint, they may have recovered their memory and recovered reasonably completley, and yet be advised not to drive a car. One reason is, of course, that if he gets into an accident—even though it may not be his fault—a jury might feel that he is to blame or an insurance company may rule against him and it might be difficult to enforce his claim. I have had this occur in several of my own cases.

It was surprising how safely patients were able to drive themselves to and from the office for some of their treatments even while in the midst of organic confusion. I did, of course, interdict such driving and always instructed their relatives to see that they were conveyed to the office and home without their driving the car, but I found out later that they persisted in driving. On the other hand, we have had patients who were

unable to find their way home even when put in a taxi with specific instructions to the driver.

Conclusion

From the above, we are able to bring these points to the attention of lawmakers and others concerned with medico-legal matters:

1. Attitudes concerning the law need to be revised in terms of new treatments, and the need for quick treatment, and toward the personality changes due to the drastic treatment a patient has had.
2. Since crimes may be committed by mentally sick people who should be properly treated, early treatment may be necessary and the matter of by-passing all criminal procedures in such cases might well be looked into.
3. The problem of amnesia due to shock treatment is a very important one. It makes it impossible for some defendants to give testimony in their own defense and it might well invalidate the testimony of many witnesses or invalidate the judicial picture in the case of a juror or judge who has had shock treatment, or is potentially in need of it.
4. The medico-legal problem of protecting the rights of persons whose judgment is poor about what they should have in the way of psychiatric treatment needs revision. It is necessary to see that they are given such treatment—perhaps by force—contrary to many current statutes. The right of the person to refuse surgery when he is of sound mind is one thing. The right to refuse psychiatric treatment, including shock treatment, when he is not of sound mind, is something else.

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Symposium on Problems of Alcohol and Intoxication*

Participants: *R. N. Harger, Ph.D.; Herman A. Heise, M.D.; Charles O. Sturdevant, M.D.; Fred E. Inbau, B.S., LL.M.; George E. Hall, J.D.*

Distribution of Absorbed Alcohol; Body Materials Which Yield Reliable Results

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The first person to propose a chemical test for intoxication was E. M. P. Widmark, Professor of Medical Biochemistry at the University of Lund in Sweden. In 1914 he published a paper (1) suggesting the determination of alcohol in urine to help take the guesswork out of cases of alleged drunken driving. Eight years later Widmark reported his well-known micro method for blood alcohol (2) which has been widely used in Europe.

Speed of absorption of alcohol

This is a very rapid process because: the molecule is small, it does not have to be digested, and it is highly soluble in water. Experiments in our laboratory (3) with 41 dogs, sacrificed at intervals after receiving 1 to 6 gm. of alcohol per kg. by stomach tube, showed the following ranges in the per cent of alcohol absorbed: 15 min., 45 to 67; ½ hr., 45 to 99; 1 hr., 73 to 97; 2 hrs., 90 to 99; and 3 hrs., 97 to 100. The average per cent of absorption in ½ hr. was 94 for 0.5 gm. per kg. and 51 for 6.0 gm. per kg. For a 150 lb. person these two doses would be equivalent to about 3, and 36, fluid ounces of 100-proof whisky.

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Correlation between the level of alcohol in brain with that of blood, liver and muscle

Fifty-three dogs were used by us (3) to study the distribution of absorbed alcohol. The time interval for 38 of the dogs was from 1 to 12 hrs., permitting storage equilibrium in most of these animals. Here the blood/brain alcohol ratio was 1.17 ± 0.13 (wt./wt.) ** the extremes being 0.82 and 1.47. For the remaining 15 dogs the time interval was ¼ hr., or ½ hr., and the blood/brain alcohol ratio was 1.03 ± 0.12. The blood samples from all 53 dogs were drawn from the saphenous vein. We believe this accounts for the lower blood/brain ratio of the dogs sacrificed during active absorption (see later discussion). The liver/brain alcohol ratio was 0.91 ± 0.07.

Due to the relatively poor circulation of blood to the voluntary muscles of a resting animal, there is a lag of about an hour before muscle tissue reaches equilibrium in the storage of orally administered alcohol. Thus, in the 12 dogs sacrificed ¼ to 1 hr. after receiving, orally, 1 to 6 gm. of alcohol per kg., the muscle/brain alcohol ratio ranged from 0.32 to 0.75, averaging 0.57; while the 12 dogs, which received 3 gm. of alcohol per kg. and were killed 3 hrs., or 12 hrs., afterward, had an average ratio of 0.92. Four dogs receiving 0.5 gm. of alcohol per kg. and killed at ½ hr. had ratios of 0.81 to 0.89.

Alcohol is about 25 times as soluble in water as in fat, and it is practically insoluble in protein and in bone minerals. When equilibrium storage has been reached the level of alcohol in each tissue is nearly proportional to the water content of the particular part. We (3) found the following equilibrium ratios for alcohol per gm. of water, with brain = 1: blood, 1.18 ± 0.08; liver, 0.94 ± 0.04; muscle, 1.01 ± 0.04; stomach tissue, 0.93 ± 0.06; upper gut, 0.97 ± 0.03; and lower gut, 0.99 ± 0.05. Intravenous, and oral, administration gave practically identical results. Nicloux (4) has suggested that the higher ratio in blood is caused by less "bound water" in it, as compared with more solid tissues.

** In the paper quoted (3) blood alcohol figures are in mgs. per ml. To estimate mgs. per gm., they were divided by 1.054. In this paper the ± figures indicate *standard deviation*.

Blood source and alcohol level

It is frequently assumed that, at a given instant, the concentration of alcohol in the blood is identical in all parts of the circulatory system. While this is essentially correct after storage equilibrium has occurred, it is not true while active absorption is in progress. During the absorption period the alcohol level in the portal blood is much higher than in the rest of the circulatory system. A somewhat lower level of alcohol is found in the arterial blood and in the blood of the right or left heart, in which locations the level is practically the same. With organs having a rich blood supply, such as the brain, lungs and kidneys, there is usually only a slight drop in the alcohol of the blood passing through the organ. On the other hand, in voluntary muscles the circulation per gram of tissue is much less than that of brain, lungs or kidneys. As a result, the venous return from muscle has a lower alcohol level than that of the arterial supply until storage equilibrium has been established.

We (5) gave 17 dogs 1 to 4 gm. of alcohol per kg. by stomach tube and, at intervals of ¼ to 3 hrs., took blood samples simultaneously from the saphenous vein and from the heart. At 15 min. the S./H. alcohol ratio was 0.67 to 0.93, ave. 0.83. At 30 min. the ratio was 0.79 to 1.02, ave. 0.91. At 1 hour, or longer, the ratio was practically unity, except for 2 one-hour tests where the saphenous vein blood still lagged by about 12 per cent.

Forney et al. (6) determined the alcohol level of brain, heart blood, and saphenous vein blood of 13 dogs decapitated 10 min. after receiving alcohol by stomach tube. The results (Fig. 1) show that the brain had reached alcohol equilibrium with the arterial blood, while there was a very marked lag in the level of peripheral venous blood.

Hulpieu and Cole (7) have further confirmed the extremely rapid uptake of alcohol by brain tissue and the slower entrance into muscle tissue. They gave rabbits 0.5 gm. of alcohol per kg., intravenously during 20 seconds, and decapitated the animals after intervals of ½ to 5 minutes. Equilibrium distribution should have resulted in a blood level of about 75 mgs. % (0.75 mg. per ml.). At ½ min. the average alcohol levels were (mgs. %): brain, 167; blood, 124; and muscle, 32;

while at 5 min. the average levels were: brain, 75; blood, 79; and muscle, 51. The sharp drop in brain and blood was due to a shift of alcohol from these areas to muscle tissue. This phenomenon also happens following rapid absorption of orally administered alcohol.

The alcohol level of capillary blood from a finger tip does not lag significantly behind the level of arterial blood, even

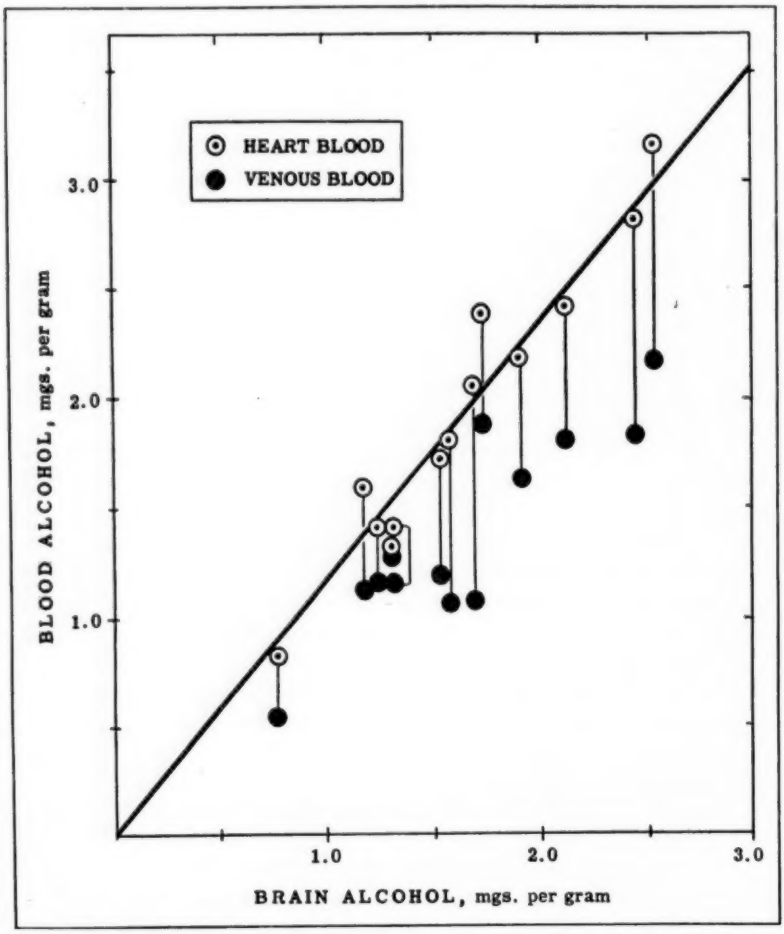

Fig. 1—Correlation between brain alcohol and alcohol in heart blood and in venous blood of 13 dogs 10 minutes after receiving 3 gm. of alcohol per kg. by stomach tube (6). The diagonal line represents a blood/brain alcohol ratio of 1.17, our average at equilibrium.

during active absorption. In 43 tests with drinking human subjects, we (8) compared the alcohol level of finger-tip capillary blood with that of cubital vein blood, drawn simultaneously. The results, shown in Fig. 2, reveal that, during the first hour after drinking, the alcohol level in peripheral venous blood is frequently 10 to 20 per cent below that of capillary blood.

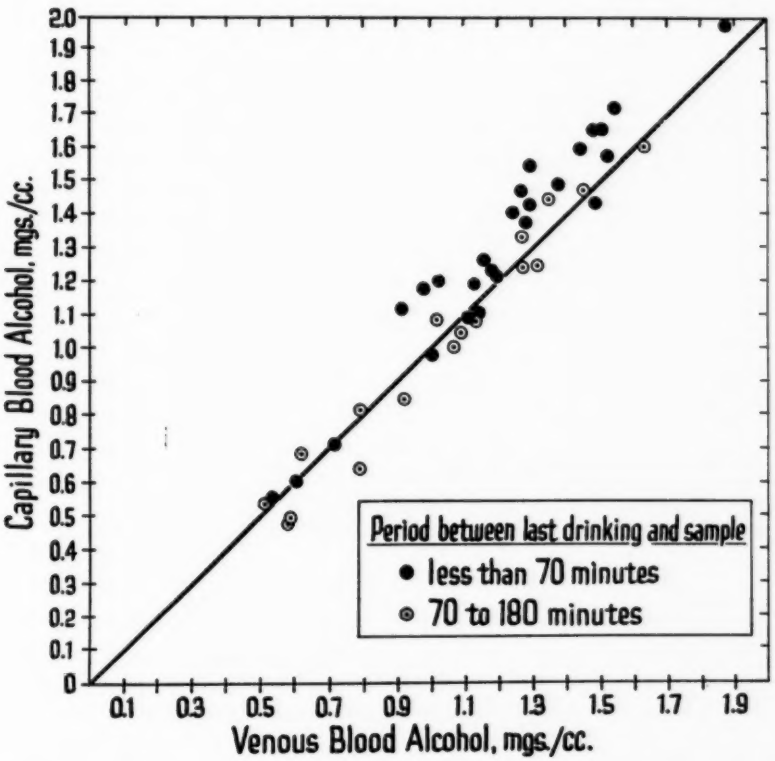

Fig. 2—Comparison of alcohol levels in venous blood and in capillary blood of human subjects (8).

Any error due to using peripheral venous blood would, of course, favor the person being tested. However, this error may explain some apparent discrepancies between blood results and blood levels calculated from breath or saliva. The reported heightened effect of alcohol during a rising blood

level may be seriously questioned where the blood samples were from a peripheral vein.

Analytical methods for blood alcohol

These are too numerous to adequately review here. Practically all yield reliable results. In most of them the alcohol is oxidized to acetic acid by a hot solution of dichromate in 10 to 20 Normal sulfuric acid. One method employs alkaline permanganate (9). Methods using the enzyme *alcohol dehydrogenase* plus diphosphopyridine nucleotode (DPN) have recently come into use (10, 11). Here, the alcohol is oxidized to acetaldehyde and the DPN goes to $DPNH_2$ which is measured photometrically at 340-70 mm. This enzyme procedure is much more specific for ethanol than are most of the chemical methods. However, the higher normal alcohols, such as propyl, butyl, etc., do give $DPNH_2$ by the enzyme method.

Urine. The passage of alcohol from blood to urine is a process of simple diffusion. An almost constant ratio exists between the concentration of alcohol in the blood and that in the urine at the time the latter is manufactured. The urine level runs about 25 per cent above that of the blood because this is about the ratio of the water content of the two fluids. Extensive studies of the correlation between the blood alcohol level and the level in urine were made by Southgate and Carter (12) and by Muehlberger and colleagues (13). If a person starts drinking with the bladder full of urine, or if he has not voided for several hours while the blood level of alcohol is decreasing, the level in the bladder urine may change more slowly than that in the blood, as the latter rises or falls. This is due to the poor blood flow through the urinary bladder wall. This discrepancy can be easily overcome by having the subject empty the bladder and then take the samples of blood and urine after waiting 15 minutes.

Saliva. The saliva/blood alcohol ratio is about 1.15. This ratio is quite constant, except for about 15 minutes after drinking, when the saliva level will exceed this ratio. Saliva/blood alcohol studies have been made by Linde (14) and Friedemann (15).

Breath. The passage of alcohol from the pulmonary blood to alveolar air is governed by Henry's Law. This means that

the alveolar air:blood alcohol ratio is constant, and that one can calculate the concentration of alcohol in one from that in the other. In 1910 Cushny measured the pulmonary excretion of acetone, chloroform, alcohols, etc. He stated (16):

> "The exhalation of volatile substances from the lungs is exactly analogous to their evaporation from solution in water, and the pulmonary cells seem to be purely passive in this process."

The distribution of alcohol between blood and alveolar air, at the temperature at which the latter leaves the mouth, is close to 2100:1. This is the ratio obtained by Liljestrand and Linde (17), and by ourselves (18). Liljestrand and Linde's breath figures during active absorption were a little too high for this ratio, but they used peripheral venous blood which would account for the variation. Haggard and Greenberg reported a different ratio, stating in 1934 (19) that it is 1150:1, and in 1941 (20) that it should be 1300:1. In 1949 the National Safety Council appointed a special subcommittee to try to reconcile these divergent views. This subcommittee, headed by Anton J. Carlson, reported as follows (21):

> "The basic principle governing the operation of the three presently used breath alcohol methods (the Drunkometer, the Intoximeter and Alcometer) is the constant ratio existing between the concentration of alcohol in the alveolar air and the blood. Available information indicates that this alveolar air-blood ratio is approximately 1:2100."

The first person to propose the analysis of breath as a test for intoxication was Emil Bogen in 1927 (22). He analyzed breath by passing it through Anstie's reagent (0.67% $K_2Cr_2O_7$ in conc. H_2SO_4) diluted with an equal volume of water and heated in boiling water. The resulting mixture of blue-green and yellow colors was compared with a series of standards prepared by using known amounts of alcohol (Strassmann, 1891 (23); Nicloux, 1896 (24)).

Our breath method (Drunkometer) was first reported in 1931 (25) and was described more fully in 1938 (26) and 1950 (18). Our object was to devise a very simple method, which would eliminate the use of boiling water, distillation, titration, etc., and which could be performed by an intelligent

police technician having little chemical training. The reagent for alcohol which we developed is made by mixing 10 ml. of 16 N sulfuric acid and 1 ml. of 0.05 N permanganate. At room temperature this reagent reacts rapidly and quantitatively with alcohol. Its ethanol equivalent is 0.169 mg. This weight of alcohol is always caught in the test, the variable being the volume of breath containing 0.169 mg. of alcohol. This reagent is not at all affected by acetone or by the breath odors from halitosis, onions, garlic, Limburger cheese, etc. We usually need a larger volume of breath than one obtains from a single exhalation by the Haldane technique for alveolar air, so we proposed the use of ordinary mixed expired air which would be simultaneously analyzed for its content of alcohol and carbon dioxide. The latter figure gives the alveolar air equivalent of the breath used to reach the end point, since Haldane and others have shown that alveolar air normally contains close to 5.5 per cent of CO_2. The weight of CO_2 in 2100 ml. of alveolar air is 190 mgs. We use the weight of CO_2 caught in the test to calculate the weight of alcohol which accompanies 190 mgs. of CO_2 in the subject's breath, which equals the alcohol in 1 ml. of his blood. What we really determine is the *alcohol-CO_2 ratio* of his breath. Table I gives a summary of our results of direct analysis of blood compared with the calculated blood alcohol using the Drunkometer CO_2 method (18).

TABLE I
Accuracy of Prediction of Blood Alcohol Level from Analysis of Breath

Blood-Breath Correlation	Michigan State Study (21)			Drunkometer (Harger)	
	Alcometer	Intoximeter	Drunkometer	Alcohol-CO_2 Ratio 1950 (18)	Rebreathed air 1954 (8)
	% of tests	% of tests	% of tests	% of tests	% of tests
Within 5%	32	45	23	34	54
Within 10%	76	77	65	63	87
Within 15%	92	86	79	80	98
In excess of 15%	8	14	21	20	2

The volume of breath used is also measured. At first we used this result only as a rough preliminary check. However, we later found that there is a fairly constant proportion

of alveolar air in the breath sample as collected, where the subject inflates the balloon to a volume of about 2 liters. The volume reading thus gives a second answer that is quite satisfactory (18).

Rebreathed air soon becomes alveolar as regards CO_2 (27) and alcohol (20). Our 1950 paper (18) contained a few results confirming this for alcohol. In this paper we mentioned plans for further study with rebreathed air, since its use would eliminate the CO_2 determination. We conducted such a study during 1954 (8). The rebreathing was performed with a two-liter polyethylene bag. Subjects too drunk to walk unaided were able to do the rebreathing satisfactorily, if properly directed. The results of these studies are summarized in Table I.

A second breath method is the Intoximeter of Forrester which was described in 1941 (28). This method also employs the alcohol-CO_2 ratio of the breath. The procedure involves two steps: (a) removal of alcohol and CO_2 from the breath by passing it through an alcohol absorbent followed by an Ascarite tube; and (b) subsequent analysis by a chemist to determine the quantities of alcohol and CO_2 caught. About 2 liters of breath are passed through a tube containing magnesium perchlorate (Dehydrite) and then through the second tube containing Ascarite. The Dehydrite absorbs alcohol and water, but no CO_2. The tubes are then closed and sent to the chemist. He re-weighs the Ascarite tube, dissolves the Dehydrite in water, distills off the alcohol, and analyzes the distillate by a conventional dichromate method. A preliminary test has been added. A portion of the breath sample is passed, at a constant rate, through a tube having a fritted glass plug which is moistened with 10 N sulfuric acid containing a little permanganate. The time required to decolorize the permanganate gives a rough approximation of the alcohol content of the breath.

The Alcometer of Greenberg was described in 1941 (29). Alveolar air is employed. The reagent for alcohol is solid iodine pentoxide, heated to about 150° C. This chemical reacts with alcohol to yield an equivalent amount of iodine. The liberated iodine then distills over into a tube of KI-starch solution. The intensity of the resulting blue starch-iodine color is read with a photoelectric device. The subject exhales as deeply as possible through a short rubber tube which con-

nects with a metal gas-sampling chamber. The operator then pushes a button and the remainder of the procedure is completely automatic. A mechanical pump forces dry, purified air through the sample chamber, then through the heated iodine pentoxide tube, and finally through the tube of starch solution. At the end of 5 minutes the operator pushes a second button and a dial on the apparatus reads the per cent of alcohol in the blood, calculated from the breath alcohol concentration by means of the 2100:1 ratio.

Descriptive drawings of the three above-mentioned breath methods have been published by Muehlberger (30).

In 1949 the National Safety Council appointed an impartial, technical committee to study the reliability of these three breath alcohol methods. This study was directed by Dr. C. W. Muehlberger, chairman of the committee. The work was done at Michigan State College under the immediate supervision of Professor Ralph F. Turner. Blood and breath analyses were run simultaneously on a large number of drinking subjects. The committee report, published in 1953 stated (21):

> "Three methods of blood alcohol determination by analysis of the breath were studied, i. e., the use of the "Drunkometer," "Intoximeter" and "Alcometer." When these devices for the determination of alcohol concentration in the blood measured by analysis of the breath were used in the manner recommended by the authors, the results obtained were in close agreement (± .015%) with results obtained by direct analysis of the blood."

In Table I, I have calculated the per cent of the total tests conducted by this committee with the three breath methods which fall within certain limits of correlation with the corresponding results of direct analysis of blood. Table I also gives similar data for our 1950 Drunkometer results (18) and for our most recent results with rebreathed air (8).

A fourth breath alcohol apparatus was recently developed by Borkenstein (31), and is called a Breathalyzer. It employs alveolar air. The alcohol reagent is dichromate in 17 N sulfuric acid, contained in an ampule reaction tube and warmed by means of the heating element of a car cigar-lighter. The subject exhales deeply through a tube connected with the sampling chamber, in the same manner as with the Alcometer. On turning a valve, a weighted piston in the sampling cham-

ber descends, forcing the gas sample through the heated dichromate solution. The decrease in the yellow color of this solution is then measured by means of an ingenious photoelectric comparator developed by Borkenstein. In our 1954 rebreathed air study we invited Borkenstein to participate, using his Breathalyzer, and his results were good.

The levels of alcohol in breath and brain are both controlled by the level in arterial blood. Therefore, during active absorption, breath should be more reliable than peripheral venous blood for predicting the level of alcohol in the brain.

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Interpretation of Tests for Intoxication

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In order to interpret the results of chemical tests for alcoholic intoxication it is obvious that we must define intoxication. This condition has been considered to be present according to some authors only when there has been obvious mental confusion and/or muscular incoordination, while others have considered a person under the influence of alcohol as soon as only slight measurable neurologic deterioration can be demonstrated. Even with the generous concept of intoxication, there will be disagreement among examiners because of their interpretation of the obvious findings. The same person simultaneously examined by two doctors may be considered culpable by one and innocent by the other, depending on the doctor's criteria for intoxication. It is obvious, then, that all arguments are fruitless unless we agree as to what is meant by "under the influence." I am in favor of any definition which adequately describes this condition, and which can be used to convict the guilty and free the innocent. The same definition should apply in all states and preferably in all countries. At the present time we have many definitions of intoxication. To be guilty in California the individual must be so affected that he is unable to operate his vehicle in the

manner of an "ordinarily prudent and cautious person in full possession of his faculties." Dr. Henry Newman correctly interpreted this to mean that a person should not be penalized unless the alcohol had lowered his driving ability below that of the poorest licensed driver on the road. According to this definition it is extremely difficult to prove intoxication. The California law exonerates the superior individual who in spite of some loss of his driving ability due to alcohol is still a better driver than most ordinary drivers. On the other hand this superior Californian would not fare as well if scrutinized according to the laws of Arizona. Because of the difficulties of proving "under the influence" of alcohol, the National Safety Council and my committee of the American Medical Association have urged the adoption of this definition of the Supreme Court of Arizona:

> "The expression 'under the influence of intoxicating liquor' covers not only all the well known and easily recognized conditions and degrees of intoxication but any abnormal mental and physical condition which is the result of indulging in any degree in intoxicating liquors, and which tends to deprive him of that clearness of intellect and control of himself which he would otherwise possess. If the ability of the driver of an automobile has been lessened in the slightest degree by the use of intoxicating liquors, then the driver is deemed to be under the influence of intoxicating liquor. The mere fact that the driver has taken a drink does not place him under the ban of the statute unless such drink has some influence upon him lessening in some degree his ability to handle said automobile." Steffani v. State, 42 Pac. (2nd) 615.

When this interpretation of "under the influence" is applied to previously published papers, much of the ammunition of the attorneys for the defense proves to be duds. For example Jetter (1) has frequently been flung into our faces as evidence of the lack of agreement among experts. Jetter's subjects were adjudged intoxicated by a criterion which he himself set. In a recent article (2) he makes his position clear:

> "While only 50 per cent of cases in a group of habitual drinkers will be clinically intoxicated at a blood alcohol level of 0.15 Gm. per 100 cc., sufficient impairment of

faculties will be present in even this type of individual to make him a menace as a driver."

The chemical criteria for judging "under the influence" are based on some simple facts. Alcohol taken into the body is absorbed by the blood and becomes equally distributed in the water of the body. At the same time the alcohol is being oxidized by the liver at a fairly uniform rate. Therefore any body fluid or tissue can be tested, and even the breath adequately prophesies blood alcohol. There will, of course, be mathematical differences in the percentages of alcohol in the various fluids due to their water content and depending on the phase of absorption or oxidation, but generous chemical criteria for diagnosis have been established to prevent injustices. Thousands of experiments indicate that the degree of impairment of mental and physical functions closely parallels the per cent of alcohol in body fluids and breath. These experiments are valid only when the individual is adequately tested before and after drinking, the tests being so conducted as to prevent errors because of improvement by practice or deterioration from fatigue. However, one of the most important effects of alcohol, and usually the first to appear, are errors of judgment. This condition will not be evident by ordinary observation or even careful physical examination. We are fortunate in having tests which show definite impairment in this subclinical stage of intoxication. Goldberg (3) has recently used a device which measures the ability to detect the flicker in a light whose rate can be varied. This test parallels visual acuity so that a couple of drinks usually affect the eyesight as though a person were wearing dark glasses at twilight. The impairment as shown by the flicker test also closely parallels deterioration of driving ability, and the threshold for impairment according to Goldberg and Bjerver is between 0.03 and 0.04 per cent alcohol in the blood (3).

The National Safety Council and the American Medical Association have recommended standards to aid in the interpretation of the results of chemical tests. Three broad zones have been designated, which adequately but with extreme generosity to the defendant, interpret the degree of impairment of the person being tested. Although there is no minimal percentage at which there will be no effect from alcohol, they have set 0.05 per cent as a figure below which the person

should be exonerated. Alcohol percentages between 0.05 and 0.15 are considered as relevant evidence of "under the influence" but are not to be construed as prima facie evidence. Fifteen hundredths per cent or over is considered prima facie evidence of intoxication. The broad zones are necessary because biologic functions have natural variations. However the figures have been set so high that no injustice will be done even to the most intelligent person who can hide the obvious effects of alcohol temporarily, or the person who has a high tolerance to alcohol. In order to qualify as prima facie evidence of "under the influence" an average-sized man would have to accumulate at least 3 ounces of absolute alcohol in his body. This means that he would have to drink *more* than 3 ounces of alcohol in order to fail to oxidize the 3 ounces found. Whenever I have performed experiments to determine the effects of alcohol I have always been surprised at the large amounts of alcohol required to cause an alcohol concentration of 0.15 per cent, usually 4 ounces of absolute alcohol or more. Even more surprising are the findings in the average person suspected of being intoxicated. Their "only two bottles of beer" (about one ounce alcohol) have mysteriously shot the percentage of blood alcohol to about 0.30 per cent.

The relative value of the tests for alcohol in various body fluids and breath has been discussed by the National Safety Council's Committee on Tests for Intoxication in a recently published booklet "Evaluating Tests For Intoxication." Their findings can be accepted as authoritative and I will comment on them briefly. First of all it should be remembered that we are able to perform chemical tests with much more accuracy than is needed to measure a biologic function. The analogy is that of measuring the diameter of the bowel with a micro-caliper, when a yardstick would be adequate. In spite of interesting differences in the alcohol content of arterial, venous and capillary blood, it is extremely unlikely that a person would be considered to be drunk by testing arterial blood, and sober by testing venous blood. Blood from any of these sources adequately parallels brain alcohol when enough time has elapsed for absorption and equilibrium. The controversy regarding the testing of breath to predict blood alcohol has been happily settled. Several different methods have given results which agree for all practical purposes. The

alcohol in saliva is a filtrate from the blood and also adequately parallels blood alcohol.

The use of urine as a test material demands further scrutiny. That the urine is an adequate test material for practical purposes is evident since alcohol concentrations of the urine closely parallel mental and physical impairment; so that a trained observer can closely estimate the per cent of alcohol in the urine by simply examining the individual (5). The disadvantages of using urine as a test material are that the alcohol consumed is filtered from the blood and therefore the concentration in the bladder will be low shortly after drinking. If a person were tested at this time the low percentage in the urine might exonerate him. From a practical standpoint, however, the person suspected of being under the influence has usually done his drinking an hour or more before he is tested, giving sufficient time to give percentages of alcohol which reflect blood alcohol closely enough for legal purposes. Haggard (4) and his associates have administered large amounts of alcohol to several persons at bedtime and then tested them the following morning. Under these conditions, the morning specimen of urine revealed some alcohol although the blood test was negative. The per cent of alcohol in the urine in all of these cases was too low for prima facie evidence of intoxication. Under these conditions the spinal fluid would have shown about the same percentage of alcohol as the urine. Possibly the "hangover" is related to the retention of spinal fluid alcohol, and we must never forget that the blood has *arbitrarily* been chosen as the standard test material. Some opponents of the use of the urine as an adequate testing material claim that the bladder is impervious to alcohol. I performed several experiments which showed that high percentages of alcohol introduced directly into the bladder quickly disappeared, and that this phenomenon occurred in addition to the dilution from the alcohol-free urine from the kidneys. The rate of loss is proportional to the concentration of alcohol; being fairly rapid while the concentration exceeds 0.20 per cent and slowing greatly as the per cent approaches zero. The most surprising finding was that plastic sausage skins partly filled with solutions of alcohol and submerged in water at 37°C. lost their alcohol at about the same rate as similar solutions in the bladder. Also the alcohol containing sausage skins gained

weight, showing that water was being added to the contents of the artificial bladder while alcohol was being lost.

Recommendations

One of the best defenses for the person accused of being "under the influence" is that he was sober at the time of the event which led to the suspicion of his being intoxicated. He may introduce a witness who claims that he saw the event, usually an auto accident, and BELIEVING that his friend needed a "stimulant," handed him a big bottle of liquor which the friend drank. The accused then admits that he was intoxicated when examined but that he was not guilty of drunken driving. It is important to be able to refute or to prove the truth of the contention. This can often be done by making more than one test such as a simultaneous blood and urine test, or breath and urine, or saliva and urine. Knowing that the urine alcohol trails significant percentages of blood, breath or saliva alcohol by about half to one hour, the veracity of the witness can usually be determined. Suppose an accident occurred and a driver was examined for alcoholic influence a half hour later, at which time simultaneous breath and urine tests were made. The breath alcohol as translated into blood alcohol then indicated 0.15 per cent, while the urine alcohol was 0.20 per cent. Conclusion: The alibi of *heavy* drinking *after the event* is disproved and it is evident that he was drinking heartily at least an hour before the event. On the other hand the "after the event" alibi would have weight if the results of the chemical tests were blood alcohol 0.15 per cent, urine alcohol 0.05 per cent. Although simultaneous examinations of urine with some other body fluid is desirable, is is also important to perform two consecutive tests using the same body fluid. When urine tests only are being made it is advisable to have the accused empty his bladder, saving the specimen for analysis. Encourage him to drink about a pint of fluids (nonalcoholic) and then collect another specimen (a few drops are enough) about 15 minutes later, noting the time. Although the first specimen gives satisfactory results for practical purposes, the second specimen ruins the alibi that some attorney may have dreamed up, namely that the accused was drunk at midnight, that he went to bed and next morning did not empty his bladder, that he was sober when he had an

accident at ten o'clock but that the urine collected at 11 o'clock was that of the night before. Breath tests repeated at about 15-minute intervals will also be of value in determining the probable time of drinking. The performing of two or more tests is also valuable to refute the contention that the examination was made carelessly and not in duplicate as is sometimes recommended. If but a single specimen of urine is available the results of the chemical tests should be interpreted as follows: When the per cent of alcohol is 0.20 per cent or more the blood alcohol per cent at the same time can be stated to be *at least* 75 per cent of the urine alcohol. This puts the individual in the class of prima facie evidence of "under the influence." When the urine percentage is below 0.20 per cent the evidence is interpreted as "has been drinking" but not prima facie evidence of influence by alcohol.

Conclusion

The threshold for alcoholic influence is below 0.04 per cent alcohol in the blood; persons below 0.05 per cent are exonerated; prima facie evidence of alcoholic influence begins at 0.15 per cent; and there will be no conflict among scientists regarding the effects of alcohol when they accept the same definition of "under the influence." These tests are usually employed to simply confirm or deny that alcohol could have been responsible for the *observed abnormal behavior*. They are fair to the accused since they give him a chance to vindicate himself; and it is obvious that no one is accused of drunken driving simply because his flicker test is deteriorating.

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Psychological Effects of Alcoholism

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Statistics show that from 1940 to 1948 there were about 3,800,000 alcoholics in the United States, or about 3.952 per 100,000 of population (1). A practice so universal must serve some psychological need and consequently has been the subject of extensive psychological investigation. Many studies have been directed toward the identification of a particular personality type which might be prone to alcoholism. Yet, to date, Schaeffer (2) found no general agreement on this subject. Generally, it is believed that alcoholics comprise a heterogeneous group.

Alcohol is a central nervous system depressant and, as such, exerts its initial effects by lessening inhibitory controls. It has been called the super-ego solvent. Freud (3) said something similar when he described alcoholic intoxication as one of man's ways of rendering frustration tolerable (drowning one's sorrows). Since alcohol does weaken restraints and deaden anticipation of punishment, it might be expected to correlate it with criminality. Yet, since alcoholism seems to be a reaction to frustration, serving the purpose of reducing tension, it might be considered more of a substitute for crime than the cause. In this connection, Sutherland (4) observed the incidence of serious crimes increased in periods of depression, while drunkenness appeared to increase in periods of prosperity. He supported the idea that alcoholism is a reaction to frustration which is safer than crime and preferred by many when money is available.

The literature concerned with the physiological and psychological effects of alcohol, including psychoanalytic theory, stimulus response, psychology, and anthropology, indicates that the primary function of alcohol is to reduce anxiety. Thus it may be viewed as one of the mechanisms of defense employed by the personality. The individual exposed to extreme tensions, or whose tolerance for frustration and anxiety is low, may develop a neurotic reaction or, under some circumstances, may resort to alcohol for blotting out reality. He may use alcohol to relieve tension and thus come to rely

on it for this purpose, needing increasingly larger amounts to achieve this end. Whenever problems arise, he uses alcohol to relieve anxiety and then is inclined to blame the stress-provoking situation for his alcoholism rather than his own behavior in escaping stress. A vicious circle ensues, with progressive deterioration of individual values and a lessening ability to function appropriately in stressful situations.

Alcoholism has been shown to cause motor performance to become slower, more inaccurate and random, therefore less well adapted to the voluntary achievement of a specific end. Similarly, it causes a lowered efficiency in mental functioning in most people, even after the ingestion of very small amounts. It interferes with the learning process and with the retention of things learned. The mental association between words and figures is obstructed. Attention and concentration are reduced and one is not as versatile in directing thought along appropriate lines. Judgment and discrimination are impaired; thinking and reasoning are weakened.

The habitual use of alcohol produces increased tolerance which is believed to be a result of altered nervous-tissue response to the toxin. This enables the chronic alcoholic to function more effectively at a given alcohol concentration than before habituation. However, this difference may be due to the habitual drunkard's acquired compensation for lack of co-ordination and for weakened inhibitions. A number of clinical entities should be mentioned here to clarify the extent to which alcohol may seriously alter mental functioning.

Acute inebriation is well-known to all of us who have stayed to the end of the party. The stages of acute intoxication correlate with the level of blood alcohol. (5) A blood concentration of 10 mg. per cent will bring about a pleasant clearing of the head, and of 20 mg. per cent a feeling of physical well-being, friendliness and talkativeness, but by the time it reaches 50 mg. per cent our drunk is on top of the world, long-winded and wise, taking personal liberties and offering to lick anyone although he may have difficulty lighting his cigarette. From this point he begins to fade, so that at 100 mg. per cent he is talking to himself, drowsy, confused and fumbling. With further elevation of the blood-alcohol level, he needs help to walk, loses sphincter control, and at 300 mg. per cent he is stuporous. At 400 mg. per cent he is anesthetized, and may even die.

Equally serious are some of the clinical results of long-continued use of alcohol. Most common of these is the *tremulous, hallucinatory, delirious state.* Characteristic is the individual who is tremulous, shaking, complaining of jitters and often avidly seeking another drink with which to quiet himself. For some time he may have needed one before breakfast to pull himself together for the day. Usually he is the spree drinker, who keeps drinking and not eating until money or physical stamina (or both) are exhausted. The symptoms reach a peak about 24 hours after the last drink. Generalized bodily tremor is the most outstanding symptom, along with sweating, startle responses, and marked restlessness. Disordered sense perception may be present; bad dreams, distortion of familiar objects, misinterpretation of noises and shadows, and even auditory and visual hallucinations may occur. Usually hallucinations are fragmentary and disappear in a few days.

Acute hallucinosis might be considered a further advance of the foregoing, which affects chronic alcoholics and is usually precipitated by increased indulgence. This condition is characterized by the appearance of auditory hallucinations accompanied by marked fear reactions in a person who otherwise shows a relatively clear sensorium. The voices are reproachful, accusatory, and threatening, often showing a sexual content. They may threaten attack or some horrible punishment, and suicide to avoid impending consequences is a possibility. Visual hallucinations are less common, but delusions developing from the hallucinatory state may so frighten the individual that he may seek help from friends or the police.

Usually, this condition is transitory and clears with treatment, but the patient may settle down in a few weeks into a chronic hallucinatory state which eventually becomes recognized as a true schizophrenia.

Delirium tremens is much more serious than either of the foregoing conditions. This occurs in the person who has been drinking excessively for many years. Often it appears a few days after such an individual has been admitted to the hospital for some other condition, or has been placed in jail where he is deprived of his usual quota of alcohol. Sometimes it occurs as he is attempting to recover from a prolonged spree. It usually appears 72 to 96 hours after the last drink.

The clinical picture is that of a restless, tremulous individual, swept by waves of apprehension. Conversation is garbled and unintelligible, pupils are dilated, heart action rapid, sweating is marked, with sometimes an elevation of temperature. Mental function is profoundly affected. He is confused, disoriented, and misidentifies objects and persons. There are vivid hallucinations and delusions, with frenzied motor activity in response to these false perceptions. Visual hallucinations are most common. The affect is generally one of great fear, although amusement and euphoria and a relative indifference to the hallucinations may occur. Retention of immediate impressions is almost nonexistent, and it is difficult to hold attention.

The usual duration is from three to six days, with recovery beginning once rest can be obtained. The death rate in uncomplicated cases is from 3 to 4 per cent—higher with complications to which such patients are prone.

Epilepsy: Convulsive seizures among alcoholics may occur after years of prolonged drinking. Usually, seizures appear toward the end of a spree. They are of the grand mal type and may occur singly or in groups. They most often occur in the course of delirium tremens. When they occur in the course of a spree, the question of previous head injury arises, since post-traumatic epilepsy is often precipitated by alcoholic excess. In the absence of head injury, the electroencephalographic findings shortly after seizure, or between drinking bouts, are generally within normal limits.

Wernicke's syndrome is an infrequent complication of chronic alcoholism related to nutritional deficiency imposed by alcoholism. In this there are gross neurological signs in addition to a clouding of consciousness, apathy, and sometimes coma.

Korsakoff's psychosis is also believed to result from a combination of chronic alcoholism and nutritional deficit. The remarkable feature in this condition is the profound memory loss, which may not be apparent on superficial examination. This may go unrecognized until the inconsistencies of stories contrived to substitute for lack of memory become apparent. Recent, retentive and some remote memory is affected. Disorientation, inability to grasp the meaning of the situation or even to remember the most recent information may be apparent.

One condition which may often present medico-legal difficulties is that designated as *pathological intoxication.* In contrast to chronic alcoholism, this appears to occur as a direct effect of drinking, frequently, very small quantities of alcohol. Predominantly, it occurs in individuals who, in the absence of drinking, show psychopathic, hysterical, unstable or schizoid tendencies. The outstanding symptom is blind rage in which the patient becomes hyperactive, impulsive and aggressive. The behavior is fantastic and marked by a complete misunderstanding of the situation. Consciousness is impaired, with disorientation, and sometimes transient illusions and hallucinations. Crimes of violence may occur, or depression with attempts at suicide. Usually, there is only transitory, or perhaps no evidence of acute inebriation. Generally, the attack lasts a few hours to a day, then, after prolonged sleep the individual awakens with amnesia for the whole episode. It is understandable why crimes committed under such circumstances, with the attendant amnesia, might provoke conflicting ideas concerning responsibility.

What becomes of alcoholics? This is a question which physicians interested in therapy of alcoholics must often ask themselves, since treatment directed to the basic cause of alcoholism is so often unsatisfactory. Lemere (6) analyzed the life histories of 500 deceased alcoholics and arrived at some significant conclusions. In this group, 287 drank themselves to death; 7 per cent regained partial control of their drinking; 3 per cent were able to drink moderately again, and 29 per cent continued to have the problem of drinking throughout their lifetime. Eleven per cent quit drinking at some time before onset of terminal illness; of these, 7 per cent quit without the need of outside help; spiritual reconversion helped 3 per cent to become abstinent, while 1 per cent found that past medical help was effective. The average life expectancy of this group was 52 years. Many of this group quit drinking for prolonged periods of time only to relapse at a later date.

This statistical study does not support any therapeutic approach. Since alcoholism does seem to be a mechanism which any human personality may employ to reduce anxiety the understanding of the alcoholic must be individually determined. Hence goals in therapy must often be less than ideal

but realistically adapted to what might be achieved, as in the treatment of other ills which affect the human organism.

Summary

Alcoholism is discussed as a mechanism of defense employed by any human personality to reduce anxiety. Attention is directed to the lowered physical and mental performance of individuals habitually using this defense. The common syndromes resulting from excessive use of alcohol and the conflict concerning responsibility in pathological intoxication are briefly considered. It is pointed out that therapy must be approached with an understanding of the particular potentialities of the individual patient.

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Legal Problems

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Scientific test results need not be infallible in order to be admissible in evidence. They need only have a reasonable measure of precision in their indications, and they must be recognized as such in the scientific field to which they belong. Moreover, the courts don't necessarily need any legislation to guide them in this direction. They can find that out by having

the scientists come into court and offer testimony to the accuracy and scientific acceptance of the test.

Statutes are very helpful, of course, in that they relieve the proponent of the evidence of the responsibility of establishing its accuracy and acceptance, a process that takes up time and requires a great deal more effort. A statute settles all this when it authorizes the use of the results as evidence. It should be remembered, however, that it is not absolutely necessary to have a statute authorizing the admissibility of the test results. The courts can admit the evidence anyway.

In none of the statutes on chemical tests for intoxication does one find any provisions to the effect that the test results are conclusive; in other words, if a certain percentage of alcohol is detected in the defendant's body fluids or breath, none of these statutes say that that means this man is drunk. They use the language "prima facie evidence of intoxication." I didn't realize how misunderstood the term "prima facie" is. We lawyers become accustomed to it from the day we walk into a law school and apparently we have not been aware of the uncertainty of that particular language. I am all for completely eliminating any such statutory language because I am sure that if there is some misunderstanding among forensic scientists, certainly the layman is at an even greater loss, and subject to much more misunderstanding.

All "prima facie" means is that on its face this is so; in other words, if you find a specified percentage of alcohol, then let's act on the assumption that this man is intoxicated. However, when contrary evidence is offered by the accused, the presumption disappears and then the court or jury must consider all the evidence and determine the truth about the matter.

In no sense of the phrase does "prima facie" mean that conclusive weight or effect must be given to the chemical test results. Moreover—and here is another and very practical safeguard—even if we tried to do it, no one could force a fact-finding body, whether it be a jury or a judge without a jury, into accepting something as conclusive. Witness the Charlie Chaplin case in the State of California. When Chaplin was accused of being the father of Miss Barry's child, blood grouping tests excluded him as the father. There was no evidence to the contrary on the part of Miss Barry—no blood

grouping test results introduced on her behalf—and yet the jury found Chaplin to be the father of the child. So a court or jury cannot be forced to accept something as conclusive, and certainly not by using the expression "prima facie."

Unfortunately, though, as indicated before, the phrase "prima facie" has been subject to some misunderstanding. I will illustrate the sense in which it is used in either statutes or court decisions. In some statutes on reckless driving, it is provided that if a person is driving 60 miles an hour that fact is prima facie evidence of reckless driving. Therefore, when it is established that a motorist was driving 60 miles an hour, that fact alone *may* warrant a finding of guilt of reckless driving. But, if the accused offers evidence that at the time he was going 60 miles an hour the weather conditions were fine, the street was wide and dry, that he had a good view at the intersection, that there was no school nearby, and so on, then the court or jury charged with the responsibility of determining whether he is guilty of reckless driving certainly should take those factors into consideration and under such circumstances the defendant is not apt to be found guilty of reckless driving. Or, if he is found guilty despite such evidence of the wide, dry street, the good weather conditions generally, a good view of the intersections and so on and so forth, a reviewing court would in all probability reverse such a conviction.

Here is an example, therefore, of the fact that "prima facie" means anything but "conclusive" evidence.

Another problem that comes up with respect to chemical tests for intoxication is the legal necessity for tracing an exhibit. One may think the courts are finicky about this, but what they are doing is insisting upon the same sort of requirements that you insist upon with respect to scientific experiments.

An expert witness can't come into court and say "This vial was given to me by Officer X who got it from Dr. Y, and I understand it is the blood of the defendant here; I analyzed this blood and found 0.18% alcohol in it. In my opinion, the defendant was intoxicated." One can see quite readily the risks this would involve. The blood specimen may have been somebody else's. There may have been a mixup along the way. It may have been contaminated, etc. Consequently, the

courts insist that there be a proper tracing of the exhibit, and the proper and safe way would be to have the doctor who took the blood specimen come into court and testify, "This is the blood I took from the defendant at ——— (time) and ——— (place). I put a ——— (mark) on the container." It is then identified as the defendant's blood. Or an officer may testify that he saw the specimen taken, that the doctor handed it to him and that he brought it to the person who made the test—in other words, a complete tracing all the way through.

The point I am trying to make is that the courts do insist upon a proper tracing of the exhibit, so anyone who becomes involved in the taking of specimens of that sort ought to be aware of the court's insistence upon such a tracing, and that there is a good reason for insisting upon it.

Another problem that is of interest is the so-called "physician-patient" privilege. A number of states have a statute which renders as a privileged communication anything that the patient may tell the physician, and it is there just for the same reason that we have the attorney-client privilege to encourage confidential communications between a client and an attorney. In some states, instead of using the expression "confidential communication or communications," the statute may cover "information"; in other words, it may render confidential *any* information given by the patient to the physician. One state has gone so far as to protect against the disclosure of *any knowledge* obtained as a result of a medical examination.

In a state where a physician-patient privilege statute contains such expressions as "information" or "knowledge," gained as a result of a medical examination, it is well to have the blood, urine, or breath specimen taken from an injured party by someone other than the attending physician. Otherwise the test results of a chemical test may be considered as "information" or "knowledge" within the meaning of the statute and therefore not usable as evidence against the intoxicated patient.

The other problem that comes up in connection with chemical tests for intoxication is the matter of self-incrimination. To appreciate the legal issues involved, I think it is well to keep in mind the history and the policy behind this self-incrimination privilege. It came about as the result of a practice that prevailed in England in the 1600's whereby people

would be brought in off the street indiscriminately and put upon oath and questioned as to whether they had any heretical beliefs, heretical in the sense of entertaining any notions different from the accepted ones of the time. There was a lot of cruelty and a lot of human misery resulting from that practice, and gradually there was a sufficient amount of resentment. Ultimately the notion developed that people shouldn't be compelled to incriminate themselves, and what was meant by that was this practice of orally testifying against one's self.

The principal reason for retaining the privilege today is due to concern over the probability that if we did not require the police and the prosecution to go out and dig up their own evidence they would develop a tendency to sit back and rely upon what they could elicit from someone once he had taken the witness stand in court; in other words, cross-examine him and bring it out in that fashion, thus proving his guilt by his own statements.

If this be the history and the policy behind the self-incrimination privilege, I think the answer is quite clear when the question is raised: Does the use of a chemical test result, where the test has been made without the consent or over the objection of the defendant, violate his privilege against self-incrimination? With few exceptions, the courts have held that a specimen of blood, urine or breath can be taken even though the defendant is unconscious at the time or he specifically objects to it. It is not a violation of his privilege against self-incrimination, the reason being that when one takes blood, urine or breath, he is getting physical evidence and not something in the nature of testimonial evidence. The person is not being forced to speak out with reference to his guilt or innocence.

There has been some recent legislation that completely avoids the self-incrimination issue in chemical test cases involving motorists. New York passed a statute a couple of years ago based upon the principle that the use of the highways is a privilege accorded by the state, and that, therefore, the state can attach any reasonable conditions upon the exercise of that privilege. The New York statute provides, in effect, that anybody who exercises the privilege of using the New York highways, whether pursuant to a license issued by

the state or a grant to a nonresident to use the highways, he uses them subject to the condition that if he is reasonably suspected of driving while intoxicated, either he will consent to the giving up of the requested specimen or he will have to relinquish his license or his right to use the highways for a period of time. Consequently, any person who uses the highways in New York, whether he is a resident or nonresident, is deemed to have given his consent to submit to a chemical test whenever he has been arrested or detained for driving while intoxicated and there are reasonable grounds for believing that he was driving while intoxicated. The statute provides that if he refuses to give this specimen, he is not to be forced into giving it, but then his license to drive shall be immediately suspended; and after a hearing, if the facts are sufficiently well established to the satisfaction of the Driver License Authority, he is deprived of the privilege of driving an automobile.

Under further provisions in the statute a report must be given the accused, informing him of the results of the tests. Also, only a physician may extract the specimen of blood, although an officer can take the urine specimen or breath specimen. The statute also provides that the defendant has the right to have his own physician make additional chemical tests. There is a similar bill pending in the legislature of Indiana, and in several other states.

Yet another problem, which might be of particular interest to those residing in California is that even though the issue of self-incrimination has been settled by court decision, or by statute as they have in New York, be careful of this: if any violent methods are used in getting a specimen, then you run afoul of another constitutional right—the right to due process of law. The Rochin (1) case in California set up this additional safeguard. In that case a suspected narcotic peddler was forced to give up his stomach contents after a stomach-pumping process, and the United States Supreme Court held it was a violation of due process. Since then there have been several decisions in the field of chemical tests for intoxication where a person had been compelled to give up a specimen and the state courts have held that the Rochin case principle did not apply because no excessively violent methods were involved. All this suggests that common sense ought to be

exercised here and that one certainly should have no part in the taking of a specimen where excessive force may be required.

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Ethical Principles

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Ethical principles, or moral principles, are principles relating to the rightness or wrongness of a particular course of conduct at some particular point of time. Is there anything right or wrong about blood, breath or urine tests according to the mores under which we live today, and who is in a position to judge of that right or wrong?

In considering the second part of this question first, it may be pointed out that four somewhat overlapping groups are deeply concerned with the right or wrong of alcohol tests: (1) the person accused of intoxication, (2) the police officer who makes the accusation, (3) the technician who collects the breath or urine or the physician who draws the blood for testing purposes, (4) the public. Since we are all members of the public and since none of us is, I hope, in the position of an accused, I will eliminate two groups and try to consider our problem from the viewpoint solely of the police officer and the physician. How, then, should they determine the right or wrong of a testing program designed to help rid our highways of drinking drivers?

The question was once raised in an informal, private discussion, as to whether or not it is ethical to withdraw blood from an unconscious person and to submit the blood so drawn to a test for analysis of its alcoholic content. Is this taking an immoral or unethical advantage of an injured person?

Does it offend public morals or common decency? The problem raised by such a question might also be raised about capturing breath from an unconscious person (if that is possible), collecting urine from a secret urinal, taking fingerprints or nail parings from an unconscious person, searching his clothes for evidence of identification, and many other procedures. Some question of ethics might be raised concerning the question of narco interrogation and giving the subject a posthypnotic suggestion that he would lead police officers to the place where he had thrown the murder weapon. The common issue in all of these procedures seems to be an implication of fraud or deceit when such things are done to one who cannot consciously object.

Whether or not a particular action is ethical depends, according to the early Greeks, upon two principles of conduct. As explained by Sir R. W. Livingstone (1), a modern English philosopher, one of these principles is represented by the laws of the state forbidding an individual to do certain things and ordering him to do others. It would seem that one method of measuring the ethics of a particular act at a particular point in history would be to examine any statutes which might have been enacted with reference to that act. Our laws are certainly a reflection of the morals of our times. The other principle of conduct, again as expressed by Sir Livingstone, involves the individual as an agent free to do as he chooses. This principle has a master, or what might be roughly translated as a conscience, a sense of things that are not done, an understanding of the things a man feels inconsistent with his self-respect or his honor and consequently will not engage in, although there is no one to prevent him from doing so. The character of a person is revealed by observing what his conscience allows to be done and what it forbids.

In the field of testing the drinking driver there is no statute either specifically *requiring* or specifically *prohibiting* either a police officer or a physician from doing anything so far as his treatment of unconscious persons is concerned. Since the public has not spoken on the subject through the legislature, the policeman and the physician must, therefore, fall back upon the second guide to conduct, namely, conscience.

To assist the physician in developing his conscience, the medical profession has adopted principles of medical ethics. Similarly, a code of professional ethics for the police service has been proposed, although it has not yet been widely adopted. Neither of these sets of principles, however, specifically treat our problem. The principles of medical ethics direct physicians, as good citizens, to bear their part in enforcing the laws of the community. The code of professional ethics for the police service emphasizes the importance of respecting due process of law. These two injunctions dovetail together.

In matters relating to crime, it is the moral and social duty of a physician to render such assistance as he can in the investigation and discovery of crime and to furnish all information possible which will lead to the detection and arrest of a criminal. Sometimes a physician will hesitate to furnish needed assistance because of a fear of breaching a confidential relationship. This apprehension may be somewhat allayed, so far as the question I have posed is concerned, for three reasons: (1) Laws relating to privileged communications and confidential disclosures only apply to instances in which the physician-patient relationship exists. No such relationship usually obtains when the physician is merely assisting the law enforcement officer. (2) It is quite likely that the person from whom a blood sample is taken will benefit from the taking. It may show he was not intoxicated. If so, can it not be presumed that he would have consented to the procedure had he been given the opportunity to do so or had he been able to do so? (3) There should be no reason why the police officer and therefore the public should be denied evidence that would bring the guilty to justice. If the test shows guilt, the individual is not entitled to have his offense hidden from society because a physician fears civil suit for revealing it.

In the last analysis, both the physician and the police officer must themselves resolve the conflict of their dual allegiances, that is, their allegiance to an individual and their allegiance to society. In the absence of statutory command, it is the conscience of each which will control. If the act necessary is not offensive to public policy, if it does not offend the idea of public justice, if it is within the limits of fair play, or due

process of law, there would seem to be no reason why the physician and the police officer cannot continue to co-operate for the benefit of the public and for the proper administration of justice.

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Evaluation of Procedures Used in Determining the Cause of Unexplained and Violent Death: A Toxicologic Approach *

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This paper summarizes the results of an investigation carried out during the first six months of 1954 at the Coroner's Office of the City and County of San Francisco. The purpose of the investigation was to evaluate, insofar as was possible by means of selected toxicological tests, the thoroughness and accuracy of certain procedures necessary to the function of the Coroner's Office.

By carrying out the tests described below, we believe that an evaluation could be made of the completeness and competency of the investigations made by the deputy coroners, the validity of the autopsy surgeon's interpretation of gross autopsy findings, and the incidence of unsuspected homicides due to poisoning which might have escaped the attention of the police. During this study it was also possible to determine the frequency of detectable barbiturate blood levels in the group.

Experimental Design

Analyses for three specific groups of poisons were made on all cases which came under the Coroner's jurisdiction. The analyses consisted of search for and quantitation of barbitals, alcohol, and certain metals and metalloids. The reasons for analyzing for these particular substances were that the fre-

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quency of the first two was sufficient to permit statistical analysis, while the finding of metals and metalloid substances arouses a suspicion of homicide, since these compounds are among the poisons most frequently used for this purpose.

A comparison was made of the frequency with which these substances were found during the period of investigation, and the occurrence recorded for the first six months of the three preceding years. The specimens submitted for toxicological analysis were obtained at autopsy, and included the following tissues in the amounts stated: blood, one pint; kidney, one half (approximately 100 gm.); stomach and its content. In addition, other tissues and body fluids (liver, hair, bone, and urine) were taken when the autopsy surgeon believed a general toxicological analysis might be required. The following tests were performed in this study:

A. *Barbitals.* The method of Gould and Hine (1949) was used. The threshold of detection is 0.4 mg. % of a barbital in the quantity of sample routinely used, and the accuracy of the method is ± 10%.

B. *Alcohol.* The method of Kozelka and Hine (1941) was used. The lower limit of sensitivity was 0.005%; the accuracy was ± 5% over the usual range (0.05 to 0.5%). Levels of less than 0.01% were not reported as positive or considered to be significant.

C. *Metals and metalloids.* The modified Reinsch method as described by Gettler (1950) was used. The metals determined by this procedure are antimony, arsenic, bismuth, and mercury.

The lower level of detectability is approximately 20 micrograms for most of these substances. In all cases where positive results were obtained, specific chemical analyses were used to quantitate the material.

Results

Approximately the same number of coroner's cases was encountered during each of the four years. The number of cases which came to the toxicologist and the percentage of the total number of coroner's cases are shown in Table 1. In 1954, 1143 cases were studied. The remaining 25 cases were not analyzed, either because tissues were too badly decomposed, or because the autopsy surgeon failed to submit them.

Table 1. Number and Per Cent of Toxicological Analyses

Year *	Number of Coroner's Cases	Toxicological Analyses	
		Number	Per Cent
1954	1168	1143	97.9
1953	1205	333	27.6
1952	1229	471	38.3
1951	1219	433	35.5

* January to June, inclusive

Barbiturates. There were 74 positive barbiturate cases during the test period, an incidence of 6.33%. Of these, 34 (2.91%) were fatal cases, that is, the history, pathologic findings, and blood level were such as to indicate lethal barbiturate intoxication to the pathologist. In 40 cases (3.2%) the circumstances and barbiturate level were such that the drug was not considered contributory to death. The percentage of both positive and fatal barbiturate cases was significantly greater than has been reported in other jurisdictions (Turkel, 1953); however, while the incidence of positive cases was significantly greater, the incidence of fatal intoxication did not differ significantly from that of the preceding year. During 1954 there were 4 cases in which the presence of lethal levels of barbiturate was established through routine toxicological examination of all cases. In these cases there was neither evidence at the scene of death nor findings at autopsy that would suggest the possibility of barbiturate poisoning. While statistically this represents a small percentage of the total barbiturate deaths discovered, medicolegally each individual case is of the utmost importance.

Table 2. Number and Per Cent of Barbiturate Cases

	Total Barbiturate Cases			Fatal Barbiturate Cases			Nonfatal Barbiturate Cases		
Year *	Cases	No.	%	No.	% Total	% Barb. Cases	No.	% Total	% Barb. Cases
1954	1168	74	6.33	34	2.91	45.9	40[a]	3.42	54.1
1953	1205	36	2.09	30	2.49	83.3	6	0.50	16.7
1952	1229	23	1.87	20	1.63	86.9	3	0.24	13.0
1951	1219	26	2.13	25	2.05	96.2	1[b]	0.08	3.8

* January through June, inclusive
a Including 5 anesthetic deaths
b Anesthetic death

In our opinion, in 40 cases in which barbiturates were discovered, their presence was noncontributory to death. These cases could be divided into two groups, hospitalized and ambulatory. Included in the 17 hospital deaths were 6 occurring during anesthesia, in which pentothal sodium was detected in the blood in quantities which would not indicate death from depression of the central nervous system. Since their role in contributing to cardiac arrest was not clearly established, they were not included among the fatal barbital cases. None of the other deaths could be grouped in a diagnostic class containing more than 2 cases. Of the 24 ambulatory, nonhospitalized cases, 16 had been under a doctor's care for heart disease. The other 7 were distributed under various diagnoses such as ulcer, alcoholism, uremia, etc. In almost all of these, phenobarbital was the barbiturate identified.

In summarizing this study of barbiturate cases, we believe the following conclusions may be drawn:

1. Analysis of 98% of coroner's cases for the presence of barbiturate did not demonstrate a significant increase in the number of barbital deaths over that of the preceding year, and did not contribute to the determination of the cause of death.
2. Unless an analysis for barbiturates is made routinely, some cases of fatal barbiturate intoxication will be unrecognized.
3. In most cases where barbiturates were found, death was due to other causes.
4. Blood barbiturate levels of 0.4 mg. % or greater were found in 6.33% of the cases coming to the coroner's attention.

We do not suggest that these data determine the incidence of barbiturate habituation, since the drugs commonly used for that purpose disappear rapidly from the blood stream, and thus are not likely to be detected.

Alcohol. Alcohol determinations were made on 1083 coroners' cases (92.5%) during the test period. The principal groups not tested were those that had been confined in hospitals for more than three days, and those with no possibility of alcohol intake, e.g., newborn children.

In 15.5% of all coroner's cases (16.7% of those examined) alcohol was present in the blood. In 119 (10.2% of all coroner's cases) the levels were greater than 0.1%. While on inspection these percentages seem to differ from those encountered in preceding years, there was no significant differ-

ence according to the chi-square test (P-0.05). In only three cases was death due primarily to acute alcoholism. There were, however, two cases with very high blood alcohol levels in which the autopsy surgeon did not request an alcohol analysis. In the 116 other cases in which the blood alcohol level was greater than 0.1%, it was the pathologist's opinion that while alcohol contributed to the death of the individual, a more satisfactory primary cause of death could be found. It is the opinion of one of us (H. D. M.) that acute alcoholism does not account for death in cases where the level is less than 0.3% except in extremely rare cases, and then only by exclusion.

Table 3. Number and Per Cent of Alcohol Cases

		Analyzed for Alcohol		Positive for Alcohol			Alcohol Over 0.1%			
Year *	Total Cases	No.	%	No.	% Total	% Analyses	No.	% Total	% Anal.	Fatal Cases
1954	1168	1083	92.5	181	15.5	16.71	119	10.18	10.99	3
1953	1205	294	24.5	108	8.96	36.73	79	6.56	26.87	0
1952	1229	403	32.8	133	10.82	33.00	87	7.08	21.59	3
1951	1219	369	30.3	146	11.97	39.57	95	7.79	25.75	3

* January to June, inclusive

We will not discuss the significance of the incidence of acute alcoholism in coroner's cases in this paper; however, we believe that the 10.18% incidence of significant levels is worthy of mention.

Reinsch test. During the test period, 1143 Reinsch tests were performed. In 31 instances the metal content of the kidney was increased. Arsenic was found in 2, mercury in 28, and bismuth in 1. In no case was there evidence that the poison had been given with murderous intent. In the three fatal cases, there was evidence from the history or gross pathologic findings that heavy metal poisoning should be suspected. A comparison of the number of cases in which metals were found during the four years appears in Table 4.

Table 4. Number of Cases Positive to the Reinsch Test

Year *	Total Positive	Arsenic	Mercury	Bismuth
1954	31	2	28	1
1953	2	2	0	0
1952	4	2	2	0
1951	2	2	0	0

* January to June, inclusive

It is apparent that the incidence of poisoning by these metals and metalloids is extremely low in our cases, and that routine testing for these substances does not disclose a greater number of fatal cases. The two cases of arsenic and one of mercury were acute and fatal. In the other cases, death was from other causes and followed therapeutic administration of the agents. In no instance was metal poisoning discovered only by means of the Reinsch test.

Other toxic materials. In addition to the cases in which barbitals, alcohol and metals were found, there were a number in which other toxic materials were identified and established as the cause of death. In general, a suspicion of their presence was raised by the history or the autopsy findings. In a relatively few cases, they were uncovered during the course of a general toxicological examination.

The most frequently occurring substance was carbon monoxide. No other single substance approached the frequency of this toxic gas in causing death. The total number and number of types of agent are shown in Table 5. The lower total for 1954 is due largely to a decrease in carbon monoxide deaths.

Table 5. Number of Other Toxic Substances Identified

Year *	Total Positive	Number of Different Substances Encountered
1954	26	12
1953	44	23
1952	50	17
1951	41	12

* January to June, inclusive

No cases of murder due to administration of poison were brought to the attention of the police as a result of this study. The number of murders listed during the study periods was: in 1954, 10; in 1953, 6; in 1952, 16; and in 1951, 15. The principal means of committing these murders was shooting, stabbing, strangulation, or cerebral trauma.

Upon review of these data, it is our opinion that the investigating deputy coroners and the autopsy surgeon are performing their functions with reasonable reliability and accuracy, and that no significant increase in the number of cases of

alcohol or barbiturate intoxication is likely to follow a much more extensive examination of cases.

Summary

The presence of barbiturates was demonstrated in 6.33% of coroner's cases, and in 2.91% of cases death was due to barbiturate poisoning. In 15.5% of cases alcohol was present, and in 10.18% the level was greater than 0.1%. This investigation indicates the advisability of further study of the incidence of barbital ingestion, and the relationship of acute alcoholism to violent death.

Conclusion

The results of a comprehensive toxicological program for alcohol, barbiturates, and heavy metals, failed to disclose any significant increase in the number of cases of poisoning over the number identified in previous years.

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Seminar on Blood Group Nomenclature*

Participants: ***Fred H. Allen, Boston, Massachusetts; I. Davidsohn, Chicago, Illinois; Walter S. Glazer, Detroit, Michigan; James J. Griffitts, Miami, Florida; Roger W. Marsters, Cleveland, Ohio; S. Milton Rabson, Fort Wayne, Indiana; Leon N. Sussman (Secretary), New York; Henry Sweets, Columbia, Missouri; Lester J. Unger, New York City; Briggs J. White, Washington, D. C.; Alexander S. Wiener (Chairman), Brooklyn, N. Y.***

DR. GRIFFITTS: If I may set the tone for some discussion about the nomenclature of the various blood groups, I am a member of the bewildered public. We come into contact with physicians in our work in Miami through the Blood Bank, and in the Rh antibody studies that are carried out on pregnant women. By and large, in our community, among physicians not specializing in work on blood factors, particularly the Rh group, I would say, 75 per cent of them know only of Rh+ and Rh—, and their information stops right there. When you begin to talk about rh′ or rh″ they don't want to listen any more. They don't care to learn that much about the subject. There is also a new group of physicians who talk in terms of C and D and E for these factors.

I am a relative newcomer to the field of blood grouping and Rh typing. I was certainly a member of the bewildered public before 1946, and I spent a year or two on an assignment at the National Institutes of Health in an attempt to work out minimum standards for diagnostic reagents relating to the processing of human blood. I came into contact with

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Drs. Wiener, Levine, Diamond, Levinson, Davidsohn and others in the field, and I listened and tried to learn as well as I could.

With regard to the blood grouping serums, at that time the National Institutes of Health were trying to establish standards of potency and specificity for these diagnostic reagents. It became quite apparent that there would have to be a proper name for these products—for these diagnostic reagents—and that even though the majority of the workers in the field were using Landsteiner's nomenclature for the blood groups, there were many laboratories that were still talking in terms of Moss and Jansky nomenclatures. After a survey of the experts in the field, and writing to many people as well as visiting them, I concluded that the consensus was that an arbitrary decision would have to be made to name this product. Some wanted to call it "anti-A serum" but qualify this further by saying group B, or Group III, or whatever number applied. A somewhat arbitrary decision was made by the biologics control laboratory, which I believe was good in the long run. I think somebody could have made a case for priority, perhaps, or for some other reason to hang onto one of the other terms; but it was obvious that unless you took a definite stand, you would merely vacillate from one to the other and prolong the confusion. It is my impression now that the Moss and Jansky type of nomenclature is through. I don't hear it any more, even in the small laboratories.

With regard to the Rh factors, most of the literature which I attempted to survey when preparing myself for the position at the National Institutes of Health was American, which was strongly influenced by Dr. Wiener, and rightfully so. I actually went through the maze of this subject, it was a maze to me, and all I knew at the time I began to learn anything about the various blood antigens and antibodies was based on the nomenclature which Wiener was then in the process of developing or had developed to a fairly high degree. When the British system was introduced it was not very difficult for me to translate it, that is, to use it interchangeably with Wiener's nomenclature. As a result of fact, today I have a hard time keeping from using the two nomenclatures interchangeably.

An attempt was made in 1947 to get most of the parties who were interested and competent to speak on the subject together in Washington, in the hope that the matter would be

settled and we would be spared the confusion that had resulted from the three different nomenclatures used for the A-B-O blood groups. I believe the judges were impartial, and I believe a good deal of time was devoted to hearing details on both sides of the situation. Frankly, having had a hand in arranging the meeting, I wished that the judges would make an arbitrary decision if necessary, so that one or the other of the two nomenclatures would be discarded. I myself had no knives sharpened for either one. I merely wanted a single nomenclature, and I felt that anybody could learn either of them if he were forced to use only one. However, the judges did not feel that they could assume that responsibility. There was too genuine a disagreement, and I, for one, was gravely disappointed. Since then I speak a jargon of the two nomenclatures combined.

DR. GLAZER: I would merely like to state that in my practice in Detroit, where the public as a whole appears to be unfamiliar with the workings of this whole Rh system especially, my procedure in sending reports so far has been to use the International Rh-Hr nomenclature and to follow this by the C-D-E notations in parentheses. Similarly, when reporting the Kell type, I may say "K(Kell+)," and similarly for the others factors.

DR. WHITE: I am very much interested in all phases of blood work. In our laboratory we don't do any basic research in discovering these new blood classifications; as far as the nomenclature is concerned, I am more or less in the same position as Dr. Glazer. I am interested in seeing the most usable type of nomenclature adopted.

In connection with our work I might say that we are particularly interested in dried blood stains. If any of you gentlemen have any suggestions as to what process we could use to attack the problem of adapting these new Rh or other typings to dried blood, I certainly would appreciate it. We attempted to do that several years ago, and we obtained assistance from Dr. Diamond and Dr. Allen in Boston, and tested a number of Rh sera. I was never able to obtain results which would indicate that we could adapt those tests to dried blood.

As far as the other factors are concerned, considerable work has been done on M and N. We tried for two or three years, and tests on dried blood stains seemed to work very

well on M, but I never could get a satisfactory result from the N test. There seemed to be a sort of nonspecific absorption, as I recall. I discussed this with Dr. Wiener one time, and I believe that was about what he found.

I did some preliminary experiments with Dr. Sanger at the time she visited in Boston several years ago and came to our laboratory. We took the anti-S serum she had, and, as I recall, we tested about ten different blood specimens (the limited number we had available), and found that we were able to distinguish very readily between S+ and S— in dried blood.

DR. RABSON: I don't want to engage in a discussion of the merits of the Rh-Hr versus the C-D-E system, but merely show the point of view of the general pathologist who has the job of training a staff of technicians. In teaching, I use both systems, pointing out briefly the reasons why each system has adherents.

I find that for rapid demonstration on a blackboard the C-D-E system is better because one uses only simple letters without any qualifying symbols like prime and double prime. However, I can see how for oral communication the C-D-E system is unsatisfactory because the chances for error are so great. C sounds like D, and D sounds like E. When reporting by telephone (and we are reluctant to do that), we therefore use the Rh-Hr system, while in our written reports we use both systems.

My second point is this: An elderly physician in San Jose, California reports that erythrocyte agglutinogens are undisturbed in alcoholic solutions or extracts. I would like to know if anyone here is familiar with this. I think the strength alcohol tried was 70 to 80 per cent.

DR. GRIFFITTS: The ABO agglutinogens?

DR. RABSON: Yes. In alcoholic hemolysis of erythrocytes.

CHAIRMAN WIENER: They remain in the stromata?

DR. RABSON: Apparently they are laked out, and there is reaction still obtainable.

CHAIRMAN WIENER: With the stromata?

DR. RABSON: Yes.

DR. GRIFFITTS: The A and B substances are soluble in alcohol.

CHAIRMAN WIENER: Only very slightly. It is very disappointing how little will go into solution in alcohol. I

can well understand if you lake out the erythrocytes with alcohol, most of the A and B activity will remain in the stromata.

DR. GRIFFITTS: We have prepared some rather—well not exactly purified antigens from human cells, but partially purified, at least—and found that the A and B agglutinogens will take an awful beating—boiling saliva and that sort of thing. A chemist associated with us has offered the following speculation. Blood group substance A, for example, might be levorotatory; substance B might rotate to the right while O might be a neutral mixture of the two. Absolutely no investigation on the point has been made, but things that simple ought to be tried just once. If it happened to work that way, it certainly would be a nice thing.

DR. ALLEN: I can say one thing in regard to the effect of alcohol on antigens. When Dr. Carter in Pittsburgh first described the Rh hapten, some men in the Department of Bacteriology at Harvard Medical School attempted to confirm and repeat her work. They found however that in their treatment of blood cells with alcohol the Rh antigen appeared to be completely destroyed.

DR. MARSTERS: I thoroughly agree with Dr. Griffitt's comments on the Moss and Jansky terminologies. I think it would be simpler if we had just one nomenclature and stuck to it.

In the Cleveland area occasionally I run into technicians in some of the smaller hospitals (and some of the physicians themselves) who are still using the Moss and Jansky system, usually along with the International. I seem to recall having seen the three systems presented in the directional inserts that come around with typing serum. I think it might be a good thing if we could recommend to some of the suppliers of the typing serums that they delete the Moss and Jansky nomenclature from their directional inserts. That is probably one of the important sources of confusion.

DR. ALLEN: We might go on record as approving that suggestion. But it can be only a recommendation, I believe.

DR. RABSON: In this connection, I had the problem years ago of discouraging percentage reporting of hemoglobin. What I did first was to report percentage, with the grams in parentheses. Then I went to grams with percentages in

parentheses, and then I dropped percentages altogether. This took about six months.

In the same way I think it would be far better, should such a recommendation be made for the blood groups, that it be a progressive change rather than a sudden change which might create considerable confusion.

DR. MARSTERS: I see no point in tapering off gradually.

DR. SUSSMAN: I want to go back for a moment to the problem raised by using two systems of nomenclature. Many of the medical schools are using the C-D-E nomenclature for teaching their medical students. At our hospital we use fourth-year medical students as night and Sunday technicians; we board, house and feed them and help them through medical school in that way. Each one of them presents a challenge to us to convert them from the C-D-E, which they have been taught, to the Rh-Hr system which we use in the hospital.

It takes a short period of retraining, but a long period of association before they become glib at converting from the one table to the other—so much so, that in the Blood Bank Laboratory we have a large chart, and if there is the slightest doubt they can look at the chart. As the list of factors becomes longer, so does the chart, which looks more and more complicated. Certainly the advisability of having a single nomenclature is magnified many times by the Rh system, when you compare it with the difficulties that arose in disposing of the Moss-Jansky until we got the International nomenclature.

With Dr. Allen, we started discussion about the value of the **hr″** tests in medicolegal cases of disputed parentage. We decided it was not valid to convert information which we had concerning the factor **hr′** to what appeared to be a similar situation with the factor **hr″**. Information gained from the study of one set of antibody reactions cannot be applied to a different one without going through the entire process of making family studies all over again.

There is no basis for using past experience with a brand new factor. Thus, one may not assume the reciprocal relationship **rh″-hr″** merely by analogy to **rh′-hr′** and without supporting experimental evidence. Dr. Wiener showed in several case studies that there were people who are exceptions to this law, and he had a family (and I had a subse-

quent family) in which a person was type Rh_o and should theoretically have been **hr″** positive but was **hr″** negative. That led to considerable disturbance as to how we could apply and use this **hr″** factor in inheritance tables.

DR. UNGER: There have been several references to the A-B-O nomenclature and to the League of Nations Committee. I happened to have been a member of that Committee. We considered proposals for five different nomenclatures for the A-B-O groups, namely, the International, the Moss, the Jansky and various combinations. The use of combinations was voted down, and you can imagine what confusion would have resulted had they gone through. It took the exigencies of World War II to get rid of complications of the A-B-O nomenclature and I hope we won't have to have another World War to resolve the Rh-Hr problem.

The problem of the Rh-Hr nomenclature will have to be met head on, because there is so much confusion in the Rh-Hr system. The subject is complicated to begin with so that the nomenclature also must be correspondingly complex. I believe either we have to do as Dr. Griffitts has suggested, namely, make an arbitrary decision, or there has to be a meeting of the minds.

Dr. Wiener has pointed out that the nomenclature must take into account genes, agglutinogens and factors. A separate name is needed for each, and the designation should be understandable orally.

Doctors are individualists, and each doctor has his own ideas. Bismarck said, "Give me three Germans and you get three opinions." I paraphrase that by saying that when you have three doctors you get six opinions. And so each scientist offers his own suggestions for nomenclatures.

The use in print of italics for genes, regular type for agglutinogens, and bold face for blood factors avoids ambiguity and confusion. For example, *B* printed in italics refers to the *B* gene, B in ordinary type refers to the B agglutinogen, while **B** in boldface refers to the blood factor **B**.

An agglutinogen is an inheritable substance in the erythrocyte cell membrane, and each agglutinogen has certain serological properties. When you wish to identify an agglutinogen there is no need to describe every one of its properties. It would be the same as if I said to you, "I met Rabson." I don't have to go on and say he is an M. D. and he has retained

his hair through the years, nor tell you the color of his eyes and the shape of his nose and describe all his features. If you want to describe him more fully, you might say, "Which Rabson?" "S. M. Rabson." Thus, a name should identify, not describe. If you try to describe an Rh agglutinogen with the letters C-D-E-F, using big and little letters, your tongue gets tangled. Even when considering the A-B-O system one is apt to think it is very simple, whereas in reality it is complicated. For example, consider group A, subgroup A_1. The corresponding agglutinogen A_1 has at least four factors, four serological properties—common **A**, the **A_1**, the **F_A** and **C**. I don't think anyone here would want to change the symbol for aggultinogen A_1 to "Common A, A_1 F_A, C"; yet when one says "A_1" that signifies a particular agglutinogen having at least the four serologic characteristics **A**, **A_1**, **F_A**, and **C**.

Similarly for subgroup A_2. It is not necessary or desirable to say "Common A, O, F_A, and C." When one says or writes "A_2," that indicates an agglutinogen which has the four serological properties **A**, **O**, **F_A**, and **C**, and probably others in addition.

The same applies to agglutinogen B. This agglutinogen also has multiple factors, **Bi**, **Bii**, **Biii**, and **C**.

If we could get away from the misconception that a factor and an agglutinogen are identical and avoid using the terms interchangeably, I believe we would avoid many fallacies.

CHAIRMAN WIENER: Dr. Allen referred to the curious anomaly that there are some type Rh_o bloods (Rh_o being defined as an agglutinogen having the **Rh_o** factor, not **rh′** or **rh″**), which lack factor **hr″** or both **hr′** and **hr″** together.

As indicated by the symbols, **rh′-hr′** and **rh″-hr″** are each contrasting pairs of serological properties analogous to M and N. Based on that concept, we exclude paternity when the putative father is rh′ negative and the child is hr′ negative or when the man is hr′ negative and the child is rh′ negative and similarly for the rh″-hr″ factors.

The fact remains that rare bloods exist which lack both members of a pair of these related factors, and this creates an uneasy situation in that such exclusions of paternity cannot be considered absolute. It so happens that in family studies we have never found a contradiction to the theory involving factors **rh′** and **hr′**, but the work of Race, Sanger and Selwyn and the anomalous blood they found (and at least

two more have been found since) certainly indicates that such exceptions to the theory must exist. Therefore, such reports carry some qualification for the sake of scientific accuracy. This qualification is on a somewhat higher level than for a mutation.

Everybody is aware that exclusions of parentage by blood grouping tests theoretically could be invalidated by the occurrence of a mutation; but that is such a farfetched possibility, that it is never mentioned in the reports, although it will be admitted to by the expert on cross-examination.

In exclusions based on the reciprocal relationship of factors **rh′** and **hr′** my habit has been to say a slight qualification is necessary because of the existence of bloods lacking both these factors, which leaves open the theoretical possibility of exceptions to this law of inheritance. My practice has been to do so in a rather mild manner, and no repercussions have resulted to date.

No such qualification is necessary when the accused man belongs to type Rh_1Rh_1 and a child is type rh, or when a man is type rh and the child is type Rh_1Rh_1. In negroids the problem is more serious because exceptions appear to be more common. The absence of both **rh″** and **hr″** seems to be not too uncommon a situation, so I feel that such exclusions involving the **hr″** factor should not be used at all in negroids, and in whites only with a very strong qualification, not only because of the theoretical objections but also because so few heredity studies have been made.

DR. GRIFFITTS: In your postscript do you state any probabilities?

CHAIRMAN WIENER: No. I merely state it is a farfetched possibility. I try to minimize the importance of the qualification as much as possible.

DR. UNGER: Would you say it is one in 15,000?

CHAIRMAN WIENER: I think that estimate is too low. I would say less than one in 4,000 or 5,000 because that many mother-child combinations have already been studied without a single exception to the reciprocal relationship between **rh′** and **hr′**.

DR. ALLEN: Is that taking into account that those examples which have been demonstrated have all been homozygous, and that there is no test by which the heterozygote can be recognized?

CHAIRMAN WIENER: It does, because the reported homozygous individuals constitute a highly selected group. They were studied because they were in the nature of freaks. Each one might represent a series of 100,000 individuals; there is no way of knowing.

DR. SUSSMAN: Just for clarification, do you state in your report, assuming it is a white person, that there is an exclusion of paternity in such cases? At the bottom of the report do you say, "Slight qualification is necessary in order to allow for a very exceptional blood which has been reported," or something like that?

CHAIRMAN WIENER: That's right.

DR. SUSSMAN: You accept exclusions involving **hr′** factor, but not those involving factor **hr″**?

CHAIRMAN WIENER: Yes. I don't mention an **hr″** exclusion when negroids are involved. For whites, I might report it in a separate letter. This would not be in the nature of an official report.

DR. GRIFFITTS: Your purpose is not to give the defense attorney the opportunity, then, to try to weaken your testimony?

CHAIRMAN WIENER: That is correct. You would be surprised how clever some defense attorneys are. They will study the literature; they will dig these facts out. If they think you are trying to hide something, your position may be bad indeed. By showing you are not hiding anything and that you are aware of the situation, you steal their thunder.

The mutation matter is quite different. Everybody should be aware of the farfetched possibility of mutations, and nobody can accuse you of trying to conceal anything by failing to qualify your report to allow for mutations.

DR. GLAZER: Have you run into any complication in States where the statute specifically states that the results of blood grouping tests can be admitted in testimony only when a definite exclusion is made? Does your qualification take it out of that category of being a definite exclusion?

CHAIRMAN WIENER: No, I would not consider it so. As a matter of fact, every report theoretically should be qualified, because every biological law is subject to exception. I don't think the legislature intended to entertain as a qualification something that is so farfetched that it is beyond the realm of ordinary daily experience.

DR. GRIFFITTS: It is like an excessively long pregnancy, like a woman having a child a full year after the last possibility of contact, or something like that. While this is a biological variation and therefore possible, it is not very probable.

CHAIRMAN WIENER: Coming back to nomenclature, I think the decision in 1947 was a good one—at least, as good a decision as the committee could arrive at, at the time with the information available to them. As a matter of fact, leaving the problem open acted as stimulus to work in order to clarify the problem. I think my own writings during the last few years have shown that. However I believe the situation today is much clearer. In 1947 there were many facts which had not yet crystallized, but which are now quite clear.

The one Dr. Unger mentioned is an outstanding example, namely, the distinction between a blood factor and an agglutinogen. Although this is mentioned in my 1943 book, and can be found in Dr. Landsteiner's writings, it was not sharply crystallized in those older writings. Now the facts are perfectly clear and show that a blood factor and an agglutinogen are quite different. The former is an attribute while the latter is the substance. Each agglutinogen is generally characterized by multiple blood factors, so that there is not merely one blood factor corresponding to each agglutinogen. Therefore, the terms must not be used interchangeably, if errors are to be avoided.

This is one beneficial result of the controversy regarding the Rh-Hr nomenclature. If there had been no controversy, the need for clarifying this point would not have been so great. So the difference between blood factor and agglutinogen might still have remained obscure.

Also, there is need to emphasize the difference between a phenotype and a genotype. For the A-B-O and M-N systems, workers have always been accustomed to reporting phenotypes, and in medicolegal problems, when necessary, the corresponding genotypes are readily derived. For the Rh-Hr types, on the other hand, because of the erythroblastosis problem and the need to determine the father's genotype certain workers got into the habit of reporting "probable genotype" for the benefit of the clinician. This practice is to be condemned, because not infrequently the probable genotype turns out to be incorrect, causing considerable confusion.

Therefore, also for the Rh-Hr system it is important to report not probable genotypes but phenotypes. By sticking strictly to phenotypes in all reports, the possibility of error is avoided.

The nomenclature should represent the subject fairly and accurately. The purpose of a nomenclature is to present the exact facts correctly—to say in symbols what has actually been found. If the symbols misrepresent what has been found, or create an incorrect impression, the nomenclature is unsatisfactory.

For example, if the symbol implies that you found something which you haven't even tested for, that may lead to errors. Only last week I received a letter from a worker new in the field who had been reading the literature and who wanted to start work on the Rh types and wrote, "Where can I get anti-d serum? The people in this area don't have any. Do you know anybody who has?" That is an intelligent request from somebody who has just picked up the literature and read it. If one reads that a blood has been typed as cde, the implication is that anti-d serum was used as well as anti-c and anti-e, which of course is not true since no usable anti-d serum has ever been found.

Any name which can lead to a misconception or mistake is a bad name. The symbols used should be unambiguous and present the facts correctly, compactly, be easy to say, easy to write, and be universal, so if you use the symbol in articles here, it is just as readily understood by readers in Russia or Japan or anywhere in the world.

DR. ALLEN: Certainly it is impossible to legislate away confusion. However you slice it, the nomenclature of the Rh system is confusing to the average person. It isn't to you and me, perhaps, but certainly it is confusing to anyone who first starts to learn it. It will always be confusing to laboratory technicians in general, probably. So, you can't ever hope, I think, to make it simple.

The principal advantage of the C-D-E nomenclature, it seems to me, is that both in writing and speaking, there is a 1 to 1 correspondence between gene and antigen; so that for example, when one wishes to describe the fact that a blood has given a positive reaction with anti-Rh_0 or anti-D typing serum, one simply has to say, "This blood is D+." With the Rh-Hr nomenclature, this isn't as easy because there are a

number of genes which may give rise to such blood, namely R^1, R^2, R^o, and R^z. That creates a difficulty for the majority of people—not for the majority of experts, to be sure, but for the majority of ordinary people.

As far as attempting to establish a single nomenclature is concerned, by making a choice between the C-D-E and the Rh-Hr nomenclature, I believe that the time still isn't ripe for a final decision, because I am sure there is still a great deal to be learned about the Rh system. As things stand now, I would hate to give up either nomenclature. I talk to myself in terms of R^1, R^2 and I think to myself in those terms, but what I see in my mind is CDe, cDE and so on. I don't think I am alone in that respect.

An excellent point was brought up by Dr. Sussman, who says he has to have a chart handy for his night technicians. I can't get along without charts, either. While I am at a little later stage, I need charts showing the gene frequencies of all the various blood factors. There are so many facts to remember regarding the whole blood group field that you just have to have these charts in front of you all the time.

There is one difficulty about the C-D-E system which everyone admits, and that is the difficulty of speaking in terms of C's, D's and E's. You have to speak in terms of R^1's, R^2's, etc. But there is difficulty also with the oral use of the Rh-Hr symbols, because you can't speak in italics, and you can't speak in boldface, and you can't speak in Germanic letters. The whole problem is very complicated.

I would like to say something relating to paternity testing. In Massachusetts, we have no laws relating to the use of blood groups in paternity cases, but the reports are accepted by the courts. I would like to cite one of my cases which is of special interest.

The putative father typed as Rh_1Rh_2, the mother as type rh and the baby as type Rh_1Rh_2. Under New York law, I presume, this would have to be reported as an inconclusive result. I didn't have to do that. I was able to test the bloods of the father and mother and siblings of the putative father, and in this way was able to show that the putative father did not carry gene R^z.

If the putative father was the actual father the baby had to be genotype R^zr, but I was able to show that neither the accused man's father nor his mother had the gene R^z. The

accused man's father belonged to type Rh_1 and his mother to type Rh_2. The siblings did not contribute any further information, except that as far as we could tell from the blood typings, they were all probably the children of this grandfather-grandmother pair. So, I was able to report to the court that Mr. X, the accused man, could be the father of the child in question, or he could be the son of the putative Grandpa, but he could not be both.

DR. RABSON: Will you repeat the last sentence?

DR. ALLEN: Either the putative father or the child was illegitimate. There was a lot of corroborative evidence, too, but the defendant was acquitted.

CHAIRMAN WIENER: This is a very interesting case and unique, because this is the first case I know of where grandparents were tested in order to solve a paternity problem.

DR. ALLEN: Could you have done that in New York under New York law?

CHAIRMAN WIENER: If the people volunteer, you can do anything. Any sort of evidence that is relevant to the problem may be introduced as testimony. It is not that we could not do it, but we don't do it. The main purpose of the law is to empower the courts to order people involved in parentage disputes to submit to the tests. The law has little to do with the admissibility of the evidence.

DR. ALLEN: Here, paternity was not excluded by the tests; and therefore, if I interpret the New York law correctly, this would have had to be reported as not excluding paternity; is that right?

CHAIRMAN WIENER: Yes.

DR. ALLEN: Whether or not you had tested the accused man's parents?

CHAIRMAN WIENER: No. If you have other evidence that is relevant, I don't think the purpose of the law is to exclude evidence which may throw light on the problem.

If you have an unusual situation, one which was not anticipated by the law, by all means introduce the evidence. The law is not intended to prevent progress. How can the law anticipate a situation that has not yet arisen? The situation you describe is unique.

DR. ALLEN: If I may make one more remark, one of the difficulties of Dr. Wiener's nomenclature was illustrated in

some of Dr. Sussman's lantern slides yesterday. For example, in one of his families the father was type Rh_1rh, the mother Rh_2rh, and the child Rh_2Rh_0.

We all know what that means—that paternity is not excluded. However, to the person who takes things as they appear on the surface, it might appear that here is a child who has two genes, neither of which are present in either parent. It is just one of the facets of the confusion you get into, no matter how you arrange the nomenclature.

CHAIRMAN WIENER: Actually, there is no confusion if one keeps in mind the distinction between phenotypes and genotypes. Otherwise how can one explain why two group B parents can have a group O child; why the mating A X B can yield children of group A and group B, while mating M X N cannot yield children of types M or N, etc. To understand these facts, the phenotypes must be translated into the various corresponding genotypes, and the same applies to the Rh-Hr types. Any confusion that exists can be blamed on the common practice of speaking incorrectly of Rh genotyping when actually only the Rh-Hr phenotypes are determined by the tests.

DR. GRIFFITTS: I want to ask a question in regard to the case that Dr. Allen just mentioned. Does the New York law, which, as I understand it, require that in paternity proceedings the parties shall submit themselves to blood tests go so far as to require that the father, the previous generation and the siblings also submit?

CHAIRMAN WIENER: I doubt it. Only the parties to the action, and grandparents and siblings would not be considered parties to the action.

DR. GRIFFITTS: That would be entirely a voluntary matter?

CHAIRMAN WIENER: It would have to be voluntary.

DR. GRIFFITTS: I am still looking forward to the day when we shall have only one nomenclature. Maybe it is wishful thinking that one can ever get the clinician to understand the Rh-Hr types, but I am certain that if we had one nomenclature it would be a lot easier than with two.

I want to make a comment about blood transfusion. The transmission of malaria in blood transfusion is not terribly common, and yet in Miami, in the last four and a half years, we have had three instances of transmission of malaria. We

tried to trace the donors in order to determine what could have been done to prevent the transmission of that disease, and we were successful in one instance in finding the guilty party. However, I don't believe it would be humanly possible to screen that individual out as a dangerous donor. The recipient had received ten transfusions in a two-week period for what apparently was a severe depression of the bone marrow following sulfa drug administration. About three weeks after the last transfusion he began to run chills and fever; blood smears showed malarial parasites of the quartan type.

We were able to trace all the donors and careful histories were taken this time concerning malaria. In addition five thick blood films from each of the donors were examined for malarial parasites. There were two suspects among the donors. One was a returned GI who had been in the Army in New Guinea for two years in World War II. He had been off suppressive therapy for two years, but there was no clinical history of malaria and we were unable to find any parasites in his blood smears despite a rather careful search. The other was a 55-year-old Greek, a fellow born in Athens, a very intelligent man. He knew what malaria was. He stated that he had never had it. I questioned him, stating that malaria was very common in Greece, particularly at the time he lived there. He said he was aware of that, and that almost everyone in their neighborhood had had malaria except their family, and their father made a sort of ritual of passing out quinine every morning at breakfast. He came to this country 45 years ago and lived in New York, Seattle, Washington and Miami. At none of the places where he stayed was malaria common or endemic. Miami, incidentally, has never been an endemic area for malaria.

Nevertheless we found quartan parasites in that donor. He was a carrier, completely asymptomatic, but we would not have been able to exclude him, I believe, by any competent history taken at the time of examination.

I believe there is a need for protection of blood banks, not an unwarranted protection to cover up negligence, but against the small but definite calculated risk of malarial carriers among donors. I believe the same situation exists for infectious hepatitis and its transmission, because, as was pointed

out yesterday, there is a tendency on the part of patients who have, let's say, eager legal advice, to sue when complications follow a blood transfusion. I believe they should be rewarded if any negligence can be shown, but I think the transmission of jaundice by blood transfusion and the transmission of malaria, at least, are two inherent dangers for which there are no adequate tests by which carriers among blood donors can be screened out.

DR. ALLEN: I was once confronted with the problem of determining the blood group of a baby that had died and had been embalmed. I was very anxious to know whether it was group B or group O. There was no blood to be tested. If I remember rightly, we removed the stomach and other tissues which were tested at our state police laboratory in Massachusetts. They found that the baby was either an O or the result was inconclusive. Is there any tissue in an embalmed body which can be used for grouping tests?

CHAIRMAN WIENER: Yes, there is. If formalin has been used to embalm the body, the stomach or salivary glands is extracted with boiling water which at the same time gets rid of the formalin. The extract is then evaporated to dryness in a steam bath, and the residue taken up in saline solution. On this an inhibition test is carried out as with saliva.

QUESTION: Dr. Wiener, when you say "stomach," do you mean the stomach wall, or mucosa, or contents?

CHAIRMAN WIENER: Mucosa.

QUESTION: You are not talking about secretors now?

CHAIRMAN WIENER: No inhibition will be obtained if they are non-secretors.

QUESTION: You would not expect this to work in every case, then?

CHAIRMAN WIENER: No; only on secretors. Group O, of course, is always an inconclusive finding.

DR. RABSON: I think that the general medical public is so confused that now or any time in the near future would be a perfectly good time to introduce a new system, because now they are not proponents of any system. I was just thinking that if I were to suddenly discover the Rh system, the way I would approach it would be to express the types in terms of a fraction, in which the Rh factors appear as the numerator and the Hr factors as the denominator. Thus, you would

have one, two or three numbers as the denominators, for example, Rh type 12/46 where 1, 2, 3, are the Rh factors and 4, 5, 6 are the Hr factors.

In that way you would avoid the whole problem of Wiener and Race. Also, as I think of it now, you would remove it from the question of letters, which we are accustomed to associate, let's say, with the A-B-O system and all the rest.

CHAIRMAN WIENER: A nomenclature such as you describe was once proposed by Murray. At any rate this is hardly the time to introduce a new Rh-Hr nomenclature, because this would close out all the old literature and merely cause further confusion.

Moreover, I don't think it would be possible to think up a better nomenclature, because every reasonable method has pretty much been tried by now. I went through the literature and found that with the C-D-E notations as many as 25 different methods of designating the same phenotype are in use. The introduction of a new nomenclature would merely increase the confusion.

DR. RABSON: I didn't know it had already been done. I was merely trying to arrive at something which would be as simple as possible to meet the transfusion problem. It would not meet the problem created by questions of genetics or paternity.

CHAIRMAN WIENER: Any new nomenclature would not take the place of the old ones, but would merely add to them.

DR. GRIFFITTS: We are better off with two than three.

DR. SUSSMAN: We are bad enough off with two, and we would be worse off with three.

DR. MARSTERS: We have had two exclusions of paternity, or at least results that we interpreted as exclusions of paternity, in which the men involved were type rh. In both cases, we found that the infants were **hr′** negative (or little c negative). Both cases concerned white people.

In both cases, the court rendered a verdict in favor of the defendants. In view of the data that has come up now on the **rh′-hr′** deletion, I am not quite so secure in my feeling on these cases.

CHAIRMAN WIENER: The qualification does not apply when the accused man is type rh.

DR. SUSSMAN: Dr. Allen raised the question, which always bothered people, about how much easier it supposedly

is to report a patient as D+ rather than **Rho**+, when only the Rho or D factor has been tested for. However, this isn't quite correct, because by convention the patient need only be reported as Rh+ or Rh— if only the single test for factor **Rho** has been done.

On the other hand, if you use three sera, **Rho**, **rh′** and **rh″** antisera, then we have eight possibilities, and the reports then state Rho or Rh_1 or Rh_2 or Rh_1Rh_2, or rh, or rh′ or rh″ or rh′rh″, as the case may be. If we use five testing sera for **Rho**, **rh′**, **rh″**, **hr′** and **hr″**, there is a total of eighteen different phenotypes to report. For example, if we see a report which reads Rh_zRh_o, we know by definition that these blood cells have been clumped by **Rho**, **rh′** **rh″**, **hr′** and **hr″** antisera.

The increase in the number of phenotypes resulting from the use of all five antisera make necessary a corresponding change in name. For example, if we have blood of type Rh_z (or **Rho** positive, **rh′** positive and **rh″** positive) which is **hr′** negative but **hr″** positive, we indicate this by the name Rh_zRh_1. If we had the serum that somebody wrote to Dr. Wiener for, so-called anti-d (anti-**Hro**) serum, we would have even more phenotypes, because there we could separate the Rh_zRh_1 into two phenotypes R_zR_1 and R_zr', depending on its reaction with anti-**Hro** serum.

Different names are used to indicate exactly how many different kinds of sera were used in the testing.

On the other hand, with the C-D-E system a great deal of license is taken in reporting, since results are reported which haven't actually been obtained and haven't even been tested for. This may cause errors as in a medicolegal case which occurred not too long ago. A putative father was reported to be type cDΞ/cde, without any test having been done for the presence of e (**hr″**) on the basis that this statistically was the most likely genotype. Actually this proved to be incorrect, because tests with anti-e (anti-**hr″**) serum showed factor e to be absent, and the entire report had to be changed.

Dr. Griffitts raised a very interesting point regarding the danger of transmitting malaria and hepatitis by blood transfusion. In 1952, we reviewed more than 2,000 transfusions, and were able to trace all the donors that had been used in sixteen cases of virus hepatitis that had developed from three to nine months following blood transfusion. We had been using a pool of donors at the time, and one donor's name

appeared as having been donor for three of the cases of virus hepatitis. Among the sixteen cases there were many patients who had received multiple transfusions. At any rate, we had one man who we had reason to suspect was a carrier of the virus causing hepatitis. There was another man who had acted as donor for two of the patients with hepatitis, but in neither case was he the sole donor. Names of all the other donors appeared only once. We recalled the man who had acted as donor for 3 of our patients with hepatitis, but neither by history nor by laboratory tests were we able to demonstrate any evidence of liver disease.

DR. UNGER: I would like to clarify one point raised by Dr. Allen. He remarked that it is not possible to talk in terms of italics, boldface or Germanic letters. I disagree. We are learning to speak and write a new language, and there have to be conventions. When we read "etc." we are all quite accustomed to say "and so forth"; and for "i. e." we say "that is." Similarly, by convention, for symbol R^1 in italics, we don't say "Italic R^1," but gene R^1. Similarly, **Rho** in boldface is read factor **Rho** and Rh_o with a Germanic R is read "**Rho** variant."

We all admit that the subject is complicated, but it has been confused unnecessarily.

A common error is to use the International nomenclature for the phenotypes, under the pseudonym "shorthand symbols" and then treat the latter as if they represent genotypes. For example, in a recent article by a competent worker, on the heredity of the Rh types and linkage, a family with parents of phenotypes $Rh_1Rh_1 \times Rh_o$ is tabulated as $R_1R_1 \times R_or$. Then in a table, the expected types in the children are given as 50 per cent R_1R_o and 50 per cent R_1r, yet all the children proved to be only of type R_1r. This apparent paradox is due to the author's incorrect use of the names R_1R_1, R_or, R_1R_o, etc., first to represent phenotypes then to represent genotypes, and then again as phenotypes. To avoid confusion, the names for phenotypes and genotypes must be kept separate. Thus mating $Rh_1Rh_1 \times Rh_o$ actually represents four genotypic possibilities: $R^1R^1 \times R^or$, $R^1R^1 \times R^oR^o$, $R^1r' \times R^or$, and $R^1r' \times R^oR^o$, not merely the single one given in so-called "shorthand symbols" by that worker. From these

four genotype matings, the possible genotypes and corresponding phenotypes for the children can readily be derived.

CHAIRMAN WIENER: I might say that Dr. Unger stole quite a bit of my thunder. Nomenclature in any subject is not devised for a neophyte—it is devised primarily for the workers in the field. The complaint that the nomenclature isn't simple enough for the man who is going to medical school to learn is nonsense. Nomenclature merely translates the facts into symbols. To try to express complex facts with a simpler nomenclature is like trying to fit a size 5 shoe on a size 12 foot. Some workers are trying to use a nomenclature which is simpler than the facts, but there must be a 1 to 1 correspondence between the names and the facts. For example, there are three distinct things—genes, agglutinogens, and blood factors—to use the same symbol to represent all three leads to errors and paradoxes such as has just been cited by Dr. Unger.

As another example, consider a type Rh_1rh husband of a type rh expectant mother. In the C-D-E notations the man's type would be written in terms of the most likely genotype as CDe/cde, while the woman's type would be written as cde/cde. When in such a family, a child of type Rh_o, or in the C-D-E notations, type cDe/cde, is born this creates considerable confusion. The same occurs when the so-called "shorthand symbols" are used. Since, when these symbols are used, phenotypes are treated as if they were genotypes—namely, R_1r for the man, rr for the woman—in this case one would expect children of types R_1r or rr only and none of type R_or. On the other hand, by using the International nomenclature correctly, we realized that the mating $Rh_1rh \times rh$ actually represents three possibilities: $R^1r \times rr$, $R^1R^o \times rr$, and $R^or' \times rr$, and since the distinction between the phenotype and genotype is constantly borne in mind, the birth of a type Rh_o child in such a family does not come as a surprise.

Similarly, we don't say "most likely genotype AO"—we say group A. To write "most likely genotype AO" when there are two possibilities AA and AO would set a trap into which we could fall—group A is clear and correct.

Why set a trap for yourself when it is totally unnecessary to do so? By our tests we diagnose phenotypes; so let us report phenotypes not genotypes. If you wish you can report

both by making two columns, phenotypes on the left and genotypes on the right, for the benefit of the clinician.

Thus we would report, father type Rh_1Rh_1, and on the right list the possible genotypes R^1R^1 or R^1r'. For the mother we would report type rh with the single corresponding genotype rr. For the child the type would be reported as type Rh_1rh with the corresponding genotype R^1r. The only one with an uncertain genotype in that particular family would be the father, and the table would show that very clearly. We do this only for the benefit of the clinician whenever we think it will help clarify the report.

DR. MARSTERS: You could exclude the possibility of the father being genotype R^1r'.

CHAIRMAN WIENER: Not in this particular family until one had a string of children of type Rh_1rh, so that the chances of the father carrying an r' gene is practically nil, could you exclude the possibility of genotype R^1r' and then it remains only a statistical probability. On the other hand, once you had a single type rh′ child you would know for certain the father was of genotype R^1r', while you need a string of at least five to ten type Rh_1 children to exclude this genotype beyond reasonable doubt.

This is a good time to discuss Rh nomenclature again because a good deal more is known now than in 1947. Though the committee in 1947 did its best, I disagreed with the report and I immediately published an article attacking it. Nevertheless I believe the committee did remarkably well under the circumstances.

I understand from Dr. Workman and Dr. Murray that there is a possibility that they may call another meeting before long which would be a good thing. Five years have passed since the last and it is time now to apply our newer knowledge to the problem. While the nomenclature may have been adequate to express the facts known in 1948, since then new facts have come to light and it is time to see that the nomenclature is brought up to date.

DR. ALLEN: It is quite possible that this is the time to settle the nomenclature problem. On the other hand, there is the possibility of the discovery of new factors in the Rh system which will further complicate the nomenclature.

To start with, let us consider the antigen which Calender and Race named Cw. To incorporate this into the Rh-Hr

nomenclature it became necessary to add four new genes, namely, r'^{w}, R^{1w}, r_{y}^{w}, and R^{zw}, whereas only the single additional C^{w} was needed in the C-D-E system.

Similarly the Rh variant named D^{u} by Stratton added only one new symbol in the C-D-E nomenclature. Now we have the antigen f, and if there is also an antigen F, it may be found that 10 per cent of the genes *r* and R^{o} determine the presence in the cell of a character which results in agglutination by anti-F serum. It will then be necessary, in the Rh-Hr nomenclature, to again double the number of symbols in order to indicate the presence or absence, in a typing test, of positive or negative reactions with anti-F and anti-f.

There is no question that this can be done with the Rh-Hr nomenclature, and this problem is entirely apart from one's theories of how these blood types are inherited, and I personally believe the evidence tends to support Dr. Wiener's theory of the inheritance of the Rh-Hr types of multiple alleles. It has not been proved, but the weight of the evidence is in favor of his theory.

On the other hand, if one continues to double the number of symbols in the Rh-Hr nomenclature, there will be an awfully long list of symbols to memorize. One really does have to memorize them, because, for example, there is nothing in the symbol Rh_1 which indicates that the blood is agglutinated by anti-**rh′**, anti-**Rho** and anti-**hr″**. One simply has to memorize what all these Rh symbols stand for.

CHAIRMAN WIENER: If a new discovery disclosed the existence of twice the number of Rh-Hr types, this complication would have nothing to do with nomenclature. The number of types would be increased no matter what nomenclature was used. Moreover, as far as nomenclature is concerned, the situation is quite the reverse of what you state. Thus, after the discovery of blood factor **f**, which I prefer to call **hr**, the Rh-Hr symbol for gene *r* remained unchanged, while the corresponding C-D-E symbol was changed from cde to cdef. Similarly, all the names for the so-called "chromosomes" had to be changed to cDef, CDeF, etc., while the corresponding Rh-Hr symbols R^{o}, R^{1}, etc. are retained unchanged. Thus the situation is just the reverse of what you have stated. Moreover, since Fisher, Race, and the others are using the International Rh-Hr Nomenclature under the

guise of "shorthand" symbols, anyway, the points you raise merely underline the need to get rid of the duplicate unnecessary C-D-E symbols.

DR. ALLEN: Suppose an antiserum for F is found, and you find a certain proportion, perhaps 10 per cent, of agglutinogens determined by gene *r* give positive reaction with anti-F?

CHAIRMAN WIENER: Firstly, bridges should not be crossed until we come to them, and this applies also to nomenclature. Secondly, such a discovery would increase the number of "chromosomes" or genes, whichever you prefer to call them, so that C-D-E workers would have to have as many new chromosome names, cdef, cdeF, etc. as there would be Rh-Hr symbols for the allelic genes.

DR. ALLEN: I think many of you will be interested in a new statute which has been passed by the State of New Hampshire relative to the use of blood typing in paternity tests. It is a very advanced law and really looks into the future regarding the use of blood grouping tests in paternity disputes and for individual identification. I believe it was passed last summer by the New Hampshire legislature.

DR. UNGER: What is the essence of the law?

DR. ALLEN: In essence it takes into consideration that at the present time, in rare instances, one may find blood types in father, mother and baby which constitutes strong circumstantial evidence of paternity. For example, if one finds the putative father to be type rh'^{w}, and the mother type rh, and the baby is type rh'^{w}, that would be extremely strong presumptive evidence that the accused man really is the father. The New Hampshire statute takes such situations into consideration.

Also, there is the possibility that in the future new blood factors may be discovered, or that factors already known but not in use will be made more useful by better antisera. Then, in a certain number of cases, much more commonly than now, one will be able to offer strong presumptive evidence of paternity, and the courts apparently would be willing to accept such evidence.

DR. SUSSMAN: The statute to which Dr. Allen referred is listed as Chapter 126, State of New Hampshire 1953. The paragraphs of the law which are of particular interest, read as follows:

"4. Effect of Test Results. If the Court finds that the conclusions of all the experts, as disclosed by the evidence based upon the tests, are that the alleged father is not the father of the child, the question of paternity shall be resolved accordingly. If the experts disagree in their findings or conclusions, the question shall be submitted upon all the evidence. If the experts conclude that the blood tests show the possibility of the alleged father's paternity, admission of this evidence is within the discretion of the court, depending upon the infrequency of the blood type.

"5. Effect on the Presumption of Legitimacy. The presumption of legitimacy of a child born during wedlock is overcome if the court finds that the conclusions of all the experts, as disclosed by the evidence based upon the tests, show that the husband is not the father of the child.

DR. ALLEN: My other point has to do with the situation where a father is type Rh_1Rh_1 and the child is rh′ negative.

CHAIRMAN WIENER: The child with or without the **Rho** factor?

DR. ALLEN: With the **Rho** factor. In such a case, Dr. Wiener writes a mildly qualifying note at the bottom of the report, and I certainly shall do so in the future. Since Race and Sanger's report describing the rare bloods which are negative with both anti-**rh′** and anti-**hr′**, we have made it a practice, in such cases, to test the father with a pure anti-**rh′** serum, i. e., one which does not contain any **Rho** antibody. Then you might find that a father who originally typed Rh_1Rh_1 gave a negative reaction with a pure anti-**rh′** serum. This would then identify him as one of these rare people with blood lacking both factors **hr′** and **rh′** and you would not falsely exclude paternity.

That is one of the characteristics of the blood of these people—that while it lacks any activity against pure anti-**rh′** sera, it does give strongly positive reactions in saline with antiserum containing blocking **Rho** antibodies such as are present in the commonly used anti-**rh′** serums.

One more point about transfusion reactions. This was called to my attention the other day when someone I know was to go to the hospital for an operation. Seven days before the operation he donated a pint of his blood, which was saved and was given to him at the time of the operation—the safest possible kind of blood to receive, and a terrific way of increas-

ing the donor population. I am surprised that this has not been publicized more, and that more use hasn't been made of that type of transfusion.

DR. DAVIDSOHN: That has happened. Cases of accidents have been observed. People were victims of an accident, one with a very rare type of blood, and in the blood bank his original donation was found and was given to him. I know of two such cases.

DR. RABSON: There is only one problem, and that is that the surgeon always says, "I want to build up the patient." Our main problem is to keep the patient from being over-transfused. We try to dissuade the surgeon from using any blood at all, which would be the best way to solve the problem. Try to keep him from using blood for most patients who don't need blood, and who in a very few weeks could easily recoup their loss.

DR. GLAZER: Dr. Allen raised the matter of the New Hampshire law, of which I was not aware. I might state that the Act passed by New Hampshire probably was the same one which was originally introduced in Michigan just about one month ago. It had the same provisions, and it also had a clause in it stating that in divorce proceedings, the presumption of legitimacy was overcome if the blood test showed nonpaternity.

DR. ALLEN: Yes, I think that also was in the New Hampshire law.

DR. GLAZER: As I understand it, the law was drawn up by some committee which draws up uniform laws for enactment in all states.

Interestingly enough, I was called to the State legislature in Lansing by the senator who introduced this law, on very short notice. He called me Tuesday afternoon and said he was being called in by the Judiciary Committee the following afternoon to discuss his law, and he was a little bit uncertain on many things, and would like to know if I could help him or tell him what book he could read to get all the dope on it.

Among other things, I referred him to some passages in Mr. Schatkin's text, and told him that if he thought I could be of any help I would be there the following morning. He asked me to be there. In going over the law with him I pointed out those two clauses with which I thought he might

have some trouble, because the California law was exactly the same as his law except that it excluded the automatic overcoming of the presumption of legitimacy and the rare blood type clause.

I said that I, as a physician, was not in favor of introducing the circumstantial evidence in the rare blood type instance, because up to now we had regarded this as an absolute test and when we said that a paternity was excluded we were speaking with certainty. If we now were going to say that there is a possibility or a probability, due to a rare blood type, we would weaken the positiveness of our test and introduce the element of conjecture. However, I said, "See what the Committee says."

The first thing the Committee jumped on was the overcoming of the presumption of legitimacy. Senator Diggs readily consented to its exclusion from the bill. Strangely enough, after the discussion had gone on for some ten or fifteen minutes, another member of the Committee wanted to know if I would explain the rare blood type statement, introducing the evidence when paternity was not excluded but allowing it to indicate probability of paternity depending upon the rarity of the type shown.

I explained to him that personally I was not too much in favor of that being included in the law, for the reasons I have just outlined, and they also felt that from past experience blood tests had grown to be accepted by the profession and by the lawyers and judges as positive evidence. For that reason the rare blood type clause was also excluded.

Dr. Davidsohn, has such a law been passed in Illinois yet?

DR. DAVIDSOHN: It was passed by both houses of the legislature, but the Governor vetoed it. He vetoed it because he said it does not take care of the child, that the child's interests are not protected.

DR. RABSON: In Denmark, if the mother cannot in her own mind determine who the father is, and if she gives the names of several men and none of them can be excluded, they are all liable for the child's support. This has the advantage to the state that enough money is collected from one man to support the child and all the additional monies collected from putative fathers Nos. 2, 3, 4 and so on, go into the general treasury.

DR. SWEETS: I get around this nomenclature problem with doctors in this way: I have a group of doctors fresh out of medical school, whose backgrounds are quite different, so I use a printed report slip. If they want just the **Rho** reaction I call that "Rh testing," and it is stated that way on the printed report slip. Then, if they want us to go out further, we call that "Rh typing." We have a little box score for the Rh antibodies. At the bottom we simply print a table with the Rh nomenclature, and on the outside it says "C-D-E" because some of the boys have heard little about that. Every one of the items in the report slip has a footnote that explains what it is all about. I have mighty little trouble from my doctors. They seem to be able to figure out what the reports mean.

CHAIRMAN WIENER: *Addendum.* The meeting was terminated at this point without taking any definite action, since it was felt that blood group nomenclature was a scientific problem which could not be settled by vote, but only by work in the laboratory. It was felt that there was need for another fact-finding committee such as the one that prepared the report for the National Institutes of Health which was published in Science in 1948, and which was the basis for their regulations for labelling Rh typing sera. In the interval of two years which have passed since our meeting was held in 1954, a fact-finding committee has indeed been formed by the Committee on Medicolegal Problems of the American Medical Association. The committee has prepared a report in which it recommends that the C-D-E notations be discarded entirely, and that the original Rh-Hr nomenclature be used exclusively in medicolegal reports.

Forensic Pathology Slide Seminar*

Conducted by Arnold F. Strauss, M.D., Norfolk, Virginia

Case 4. ** **Diagnosis: Mustard Seed Embolism of Lung**

History: A 31-year-old white female was admitted to the hospital in shock with a history of vaginal bleeding for two days. Necrotic placental material was curetted from the uterus. Blood culture later yielded clostridium perfringens. Terminally there was hemolytic anemia and lower nephron nephrosis with death occurring two days after admission.

Case referred to Medical Examiner because of suspected abortion.

Discussion: The lung microscopically presents a severe congestion, edema with mononuclear cells and intra-alveolar hemorrhage: the typical expression of shock.

Within the pulmonary arteries there appear particles with the distinct walls of plant cells. Occasionally one sees little reaction in the vascular walls, especially where the emboli do not touch them. More often there is marked edema and sometimes fibrinoid necrosis. The arterial walls are infiltrated with mononuclears, neutrophils and a few lymphocytes. In some areas the wall is destroyed by granulation tissue. Neutrophils and mononuclears may infiltrate the emboli. Fibrin is deposited on their surface. Occasionally small foreign body giant cells fill out the spaces within the cell walls of the vegetable. (Figure 1.) The uterus also showed microscopically plant cell emboli with early organization, thrombosis and acute adventitial inflammation. Acute centrolobular necrosis was found in the liver.

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* Presented at the Annual Meeting of the American Academy of Forensic Sciences, February 25–27, 1954, Chicago, Illinois. [Cases 1–3 appeared in Volume 1, Number 1 of this journal.]

** Submitted by Dr. Russell Fisher, Chief Medical Examiner, Baltimore, Maryland.

Mustard releases a volatile oil, allylisothiocyanate (C_3H_5NCS), an active irritant. To its action the abortion was attributed.

Lovitt, (1), raised the question whether the hemolytic anemia of the patient was caused by Cl. perfringens, cultured at autopsy, or by the mustard. Instead of killing our rabbits after they have passed the pregnancy test, Dr. George J. Varecska injected them with 1 ml. mustard intravenously according to the woman's preparation. We have killed only one animal after 68 hours. It showed a marked arteritis about the emboli which were at least as numerous as in the sections taken from the patient. Cultures of the injected material were negative. None of the animals, even with subcutaneous deposits of 1 and 2 ml. of mustard developed anemia. We,

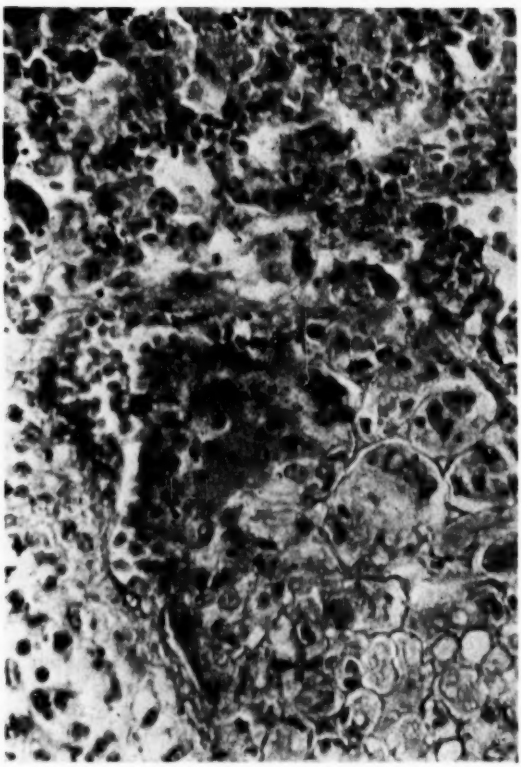

Fig. 1—Mustard seed in left lower corner. More to center foreign body giant cells within vegetable cells. Wall of vessel necrotic.

therefore, believe that Cl. perfringens must have been the cause of blood destruction, not allylisothiocyanate.

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Case 5. Diagnosis: Intestinal Pneumatosis

History: A negro male, age 46, was admitted to the hospital in a comatose state with fecal vomiting. History obtained from relatives was that the patient had suffered from periodic vomiting and had lost one hundred pounds in weight in the past three years. No history of melena or coffee-ground vomiting was elicited. He expired one hour after admission. Autopsy revealed complete intestinal obstruction by a large multicystic mass in the ileum.

Case referred to Medical Examiner because decedent was unattended during fatal illness.

Discussion: The wall of the intestine showed numerous cystic spaces in the mesentery. The septa are often torn. There is no content in the interior of these spaces. No relation to preformed tissue structures can be made out. The cysts are without epithelial lining. Their walls are fibrous and

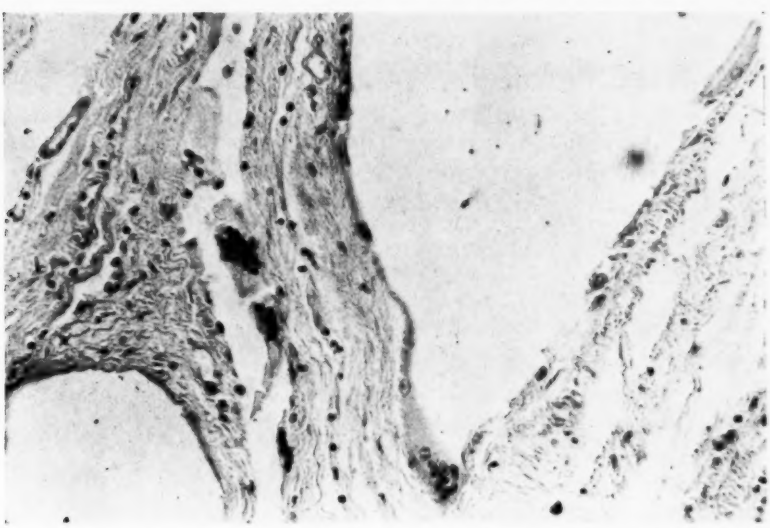

Fig. 1—Large gas-filled cysts in mesentery. The wall in the center is covered by a film of inspissated lymph in which nuclei seem to proliferate.

naked, or covered by a layer of thin spindle cells. (Figure 1.) In some areas there are flat or cuboidal cells without discernible cell boundaries or there are giant cells. (Figure 2.) Both generally have a finely granular cytoplasm. In a number of cysts the wall is covered by a pale pink staining film, likewise finely granular, in which nuclei seem to proliferate. Giant cells may look like nuclear colonies in this granular, protein or inspissated lymphlike material.

Fig. 2—Wall of left cyst is covered by a film of lymph in which a giant cell develops. In the tissue at right from center giant cells form in tissue spaces filled with lymph but free from gas.

In the intervening tissue the giant cells are often more numerous and larger. Here too, they may have a finely granular cytoplasm and no distinct boundaries. Often lymphocytic infiltration, macrophages, a few neutrophils and a rare eosinophil are present.

There is no epithelial or endothelial lining as in cystic tumors. There is no fluid content. Artefact is ruled out by the presence of vital reaction. Therefore, the diagnosis is intestinal pneumatosis.*

* This case was reported by Dr. George W. Thoma and Dr. Gordon R. Hennigar (6) from the Office of the Chief Medical Examiner and the Department of Pathology of the Medical College of Virginia.

Relevant findings at autopsy were dilation of the stomach with an estimated capacity of 4 liters, and containing 1 liter of brown foul-smelling fluid. The pylorus had a stellate scar 0.3 cm. in size. There was no obstruction at the pylorus. The cystic 20 cm. long gas-filled mass which obstructed the ileum 45 cm. above the ileocecal valve extended from the submucosa to the serosa. The intestine proximal to the mass was dilated and hypertrophic. An unusual finding in this case was the involvement of the serosa of the right diaphragm which had been adjacent to the mass.

Though rather rare in humans, pneumatosis in pigs is well-known to veterinarians (5). In children the condition is often sporadic (2) and most common during the early months of life. Any part of the gastro-intestinal tract upward from the transverse colon, most commonly the distal ileum, is found to be involved. In children the mucosa, in adults the serosa, are the preferred sites. The lesion has been observed to persist up to nine months.

Pneumatosis is full of mysteries. It is not known how the gas is formed, nor why it persists. Known facts are that the disease is commonly found with colitis, carcinoma of the stomach, obstruction, strangulated hernias, parasitic infections, passive congestion, constipation, meteorism, gastric ulcers and above all, duodenal ulcers, which are found in 45 to 75% of these cases (2, 3). A primary form exists without other diseases, perhaps after their disappearance.

In hogs pneumatosis occurs when their corn diet is reduced, and experimentally when they are fed polished rice(1).

Etiologic theories (1, 3, 5) are numerous, often explaining at least one possible causative factor.

The mechanical theory points to increased intra-intestinal gas pressure from hyperperistalsis, obstruction, coughing, sneezing and vomiting.

The bacteriologic explanations accuses nonpathogenic gas-forming organisms which are occasionally observed in the cysts. They are more commonly found in emphysematous cystitis and vaginitis. It cannot be ruled out that organisms have disappeared by the time the lesion is discovered. In one of the sections of our case I found cocci and rod-shapes which appeared to be artifically introduced.

The experience with hogs may suggest changes in the quantity of gas and of the composition of the intestinal con-

tents, and in the intestinal lining due to avitaminosis or other diseases. A change in pH liberating gas in lymphatics has been considered.

A knowledge of the nature of the gas would be helpful in better understanding of the process. None of the few analyses agrees with the other. O_2, CO_2, N and H have been found. The gas content of the intestines and air become equal by diffusion in about three hours. This and different gas contents at various ages of the lesion may be the cause of disagreement. Immediate gas collections on operative specimens would be helpful.

The causation of foreign body giant cells by gas was reported by Wright (7) who studied the effect of repeated gas injections of O_2, CO_2 and N and found a variation in cellular exudate and in giant cell formation according to the gas used. Giant cells formed the fourth day, and invariably after the tenth day. One explanation is the mechanical factor of tissue destruction varying with the number of injections and the rate of gas absorption. The microscopic sections show clearly that the giant cells arise as a reaction to inspissated tissue fluid or lymph. Moore (4) observed in his case that the few giant cells present were "in association with fibrin and fibrous deposits." Since the same giant cells occur within the tissue, away from the gas cysts, there is little need to explain them as caused by gas. The plastic film of inspissated fluid, like hyaline membranes in lungs, may be responsible for impeding the reabsorption of gas. Later the thick layer of collagen about cysts may have a similar effect. It would also explain the immobility of the gas bubbles which cannot be shifted as in tissue emphysema. They give a little cracking sound when incised, which may be caused by pressure due to reactive inflammatory contraction of their surroundings.

Gas in tissue is of forensic interest. It occurs in several conditions:

(1) In post-mortem deterioration where it is found without vital reaction, and generally in various organs.

(2) In gas bacillus infection with gangrene of tissue.

(3) In traumatic emphysema after injury to the respiratory tract and over-distention of lungs. Not uncommon is the injection of compressed air into the rectum, leading to fatal peritonitis. The gas is movable and there are no giant cells.

(4) In caisson disease with nitrogen bubbles diffusely distributed in the blood, about joints, in fat tissue, leading to myelomalacia and to infarcts at the ends of long bones.

(5) In emphysematous cystitis and vaginitis which always should cause suspicion of diabetes, and in our lesion, intestinal pneumatosis.

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Case 6. Diagnosis: Kidney in Carbontetrachloride Poisoning: Cholemic Nephrosis

History: A 29-year-old white male was admitted to the hospital with jaundice of increasing severity. He became comatose shortly thereafter, and expired with liver failure one week after admission.

Case referred to Medical Examiner because of suspected poisoning.

Discussion: The microscopic section of the kidney shows glomeruli with albuminous precipitate. Their capillaries are not anemic. The proximal convoluted tubules contain albuminous precipitate with hyalin globules. The cells are swollen and often have no brush border. Occasionally the cytoplasm seems to herniate into the lumen. Sometimes the cells have eosinophilic granular cytoplasm. Occasionally the nuclei of the cells are not stained. The descending thin limb of the loop of Henle sometimes shows vacuoles below, around and occasionally above the nucleus. These cells contain little fat. There is none in the vacuoles. In the proximal tubules are bile stained casts, the epithelial cells may be loosened from

the basement membrane. (Figure 1.) Some regeneration can be found. There is an occasional bluish staining crystal in the collecting tubules of the papillae. The medulla is congested. There are no changes of interstitial tissue or of the vessels. The pelvis shows hemorrhages.

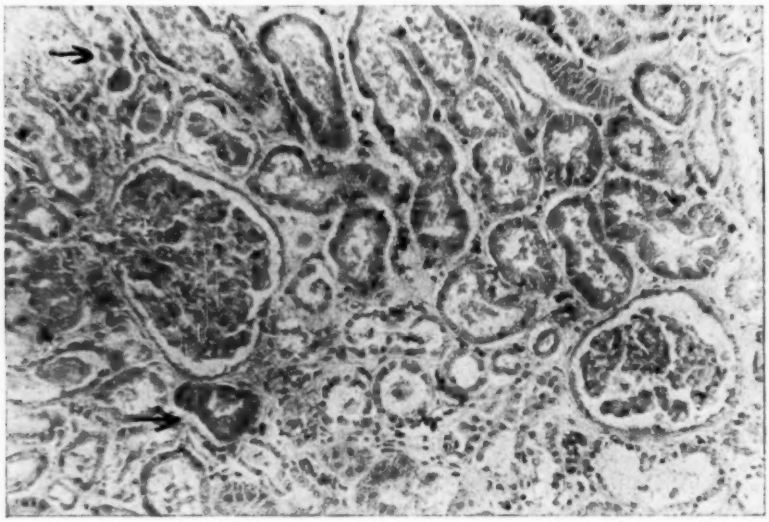

Fig. 1—Albuminous precipitate and bile stained casts (upper pointer). Hyaline droplet degeneration (lower pointer).

There is no anemia of the glomeruli, no heme-pigment or heme-casts. The basement membranes are intact. There are no signs of tubulorhexis or tubulo-venous thrombosis. There is no tubular dilatation, nor is there inflammation of the interstitial tissue.

I believe all we can say about this kidney is that the patient had albuminuria, jaundice and hematuria from the petechiae of the pelvis due to hepatic failure. We do not see severe renal changes and it is difficult to say how much of what we observe is caused by deterioration. A specific diagnosis seems to me impossible beyond saying: kidney in case of liver injury. The patient had carbontetrachloride poisoning with one plus albuminuria, hematuria and the specific gravity was 1.019. Urinary output was good. BUN was 25 and 18 mg. %.

Before discussing the renal effect of CCl_4 it seems worthwhile to look at the kidney from a morpho-forensic viewpoint.

Lucké (5) created a morphologic entity, the "lower nephron nephrosis," caused, to use Homer Smith's words, by a "myriad of contrasting dynamic mechanisms": Poisons, hemorrhage, crush injuries, transfusion accidents, overwhelming infections. Then McManus (6) proved that the upper nephron is devoid of alkaline phosphatase and, therefore, likewise damaged which was compatible with renal functional studies. Bull (2) and his co-workers in 1950 suggested, from functional studies, that there exist two distinct tubular lesions in poisoning, those from the effect of the poison on the epithelium, and those due to renal ischemia from shock. Oliver (8), in dissecting whole nephrons, did find two morphologic equivalents in his studies of mercurichloride kidneys:

(1) The proximal tubule, the principal absorbing segment, was damaged by poison causing a nephrotoxic lesion.

(2) There was disruption of the continuity of the basal membrane: tubulorhexis. This lesion on dissection occurred randomly from the origin of the tubule to its end in the collecting tubules, corresponding to tubulo-venous thrombosis and the resulting interstitial inflammation (3). This lesion is due to irregular blood supply (2, 10). Oliver related morphologic changes to functional disturbances more fully than it had ever been done. The accompanying table is taken from his publication in the *American Journal of Medicine,* October, 1953. We should make a clear distinction between the nonspecific picture of shock and the specific nephrotoxic lesions. The former may be superimposed upon the latter or either one may be found independently. When the effect of a poison is studied experimentally, shock should be avoided, if possible, to clarify the specific nephrotoxic lesion.

To illustrate the application of the principle, a short discussion of nephrotoxic lesions may be of interest. The proximal convoluted tubule shows hyalin droplet degeneration and marked eosinophilic necrosis in poisoning with mercury chloride, potassium chloride and potassium bromide poisoning, the neutralizer of cold wave lotion (1). In mercury chloride there is often calcification, and in potassium chlorate formation of methemoglobin. Uranium nitrate, cantharidine and tartrates show likewise proximal tubular lesions (5). Oxalate and the antifreeze glycol have an identical effect. They produce changes in the upper nephron with light yellow

sheaves of prismatic or rhomboid, birefringent crystals of calcium oxalate (1).

Diethylenglycol causes coarse, destructive vacuolization with extrusion of cellular cytoplasmatic contents, resembling the renal lesions of colitis and cholemia. There are often pyknotic nuclei as we saw in our kidney of lead poisoning, and there is often bilateral cortical necrosis (1). Leucine-like crystals, common in all hepato-renal lesions, are present. The punched out vacuolar changes of the central third of the liver lobules aid in diagnosis (1).

Cholemia in which we may be dealing in certain cases of carbon tetrachloride poisoning produces milder epithelial changes, vacuoli in nature, and coarse, large, irregular vacuoles which appear scooped out of the epithelium of the proximal tubule and the descending limb. These changes are also seen in severe diarrhea and colitis (1).

Hypertonic solutions of sucrose, and other sugars, as well as Asiatic cholera produce diffuse hydropic changes with the nuclei at the base of the cells and a sieve-like clearing of the cytoplasm. These changes are seen in the proximal loop and the thin descending loop of Henle (1). (Figure 2.)

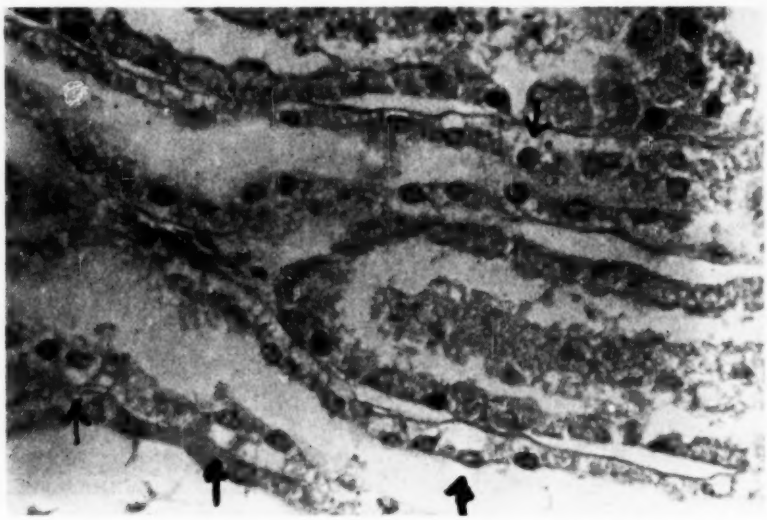

Fig. 2—Subnuclear vacuoles in descending loop of Henle.

The ascending loop is damaged mainly by phosphoric and uric acid (4).

Fatty changes may be due to phosphorus, chloroform, hallogenated hydrocarbons, ethane, bromoform, chloral (1) and to arsenic, phosgen, sulfuric acid, acetic acid, trinitrotoluol, salicylic acid, carbolic acid, perubalsam, opium and chronic morphine poisoning (9). In most volatile poisons, no direct relation between liver and kidney fat exists. In carbontetrachloride poisoning the relation is inversed (7). In chloroform poisoning a direct relation in the quantity of fat in kidney and liver is found. In lipoid nephrosis there is fat also in the glomeruli and the interstitial tissue. It is diffusely distributed throughout the cell. Anisotropic fat is seen here as well as in amyloid and in intercapillary glomerular nephrosis (1).

Under certain conditions the shock kidney may have specific features. Hydroquinone (1) will cause hemolyzed blood casts rather than hemecasts throughout the nephron. Sulfacrystals may allow a specific diagnosis.

The importance of renal changes in carbontetrachloride poisoning was emphasized by Smetana (11) who pointed out that the symptoms of hepatic damage are soon overshadowed by renal impairment. Apparently, if the patient lives long enough, cholemic nephrosis is followed by the tubular necrosis of the shock kidney. When one looks over published cases of carbontetrachloride poisoning, one gains the impression that liver damage is often more severe in ingestion cases, and kidney damage in inhalation cases where the liver does not act as a filter.

The knowledge of morphotoxology becomes a valuable weapon in the hands of the forensic pathologist when he is dealing with embalmed bodies or with poisons which are rapidly excreted or metabolized, even though the definite answer can only be given by chemistry. In hepato-renal syndrome carbontetrachloride should be suspected. Poisoning with carbontetrachloride may be accidental, occasionally suicidal. It is practically never a tool of murder.

CORRELATIONS OF STRUCTURE AND FUNCTION IN ACUTE RENAL FAILURE (ACUTE TUBULAR NECROSIS)

Functional Disturbances	Structural Changes
Etiologic Factors	
1. Functional evidence of dual etiology—ischemia and renal poison	1. Two types of tubular lesion—tubulorhexic and nephrotoxic 2. Visual demonstration of focal ischemia in renal poisoning
The Oliguric Phase	
1. Reduction in RBF and GF 2. Albuminuria 3. Hemoglobinuria 4. Loss of proximal tubule function; inability to concentrate urea and creatinine, to conserve electrolyte and to extract PAH 5. Relative maintenance of glucose Tm 6. Persistence of reduced RBF after initial insult	1. Glomerular capillaries empty with tufts collapsed but intact 2. Protein in capsular space 3. Heme pigments in capsular space and tubule lumens, heme casts 4. Beginning tubular damage—lack of proximal absorption of heme pigments increasing to tubulorhexic and nephrotoxic necrosis 5. Structural integrity of first one-third proximal convolution 6. Tubular leakage, interstitial edema, and reaction and increased intrarenal tension
The Early Diuretic Phase **Occurrence of Diuresis as Result of Reparative Phenomena**	
1. Onset of diuresis as a result of absorption of interstitial edema, increasing blood flow and filtration with continuing depression of tubular absorption of water 2. Persisting evidence of abolition of proximal tubule function (lack of ability to concentrate urea in urine, inability to conserve electrolyte or extract PAH)	1. Distended glomerular capillaries and normal appearing tufts; persisting tubular damage, nephrotoxic and tubulorhexic, with especial involvement of proximal convolution 2. Extensive atypical regeneration of renal epithelium—absence of mitochondria and consequent reduced enzymatic content of new renal epithelium; excess epithelial proliferation and tubular dilatation
The Late Diuretic Phase	
1. Gradual recovery over weeks or months of tubular functional ability with possible eventual clinical recovery. 2. Renal function after clinical recovery adequate but remaining below lower limits of normal	1. Slowly progressive maturation of regenerated tubular epithelium with ultimate reconstitution of original structures including mitochondrial organelles with their enzymatic content 2. Loss of nephrons from irreparable tubulorhexic damage and associated hypertrophy and hyperplasia of others

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Case 7. Diagnosis: So-called Interstitial Pneumonia

History: An 8-month-old white female had been in good health with no history of illness. Twenty-four to forty-eight hours before death family noted slight nasal discharge, but no fever. The child was placed in bed and was found dead the following morning.

Case referred to Medical Examiner because of unexplained unattended death.

Discussion: The sad story of a healthy appearing baby, or one with a slight cold found dead in his bed is only too familiar to the medical examiner.

Our case shows the lung congested, with edema, and spotty atelectasis. Some of the large bronchi contain a little aspirated material and bacteria with some desquamation of the epithelium, and slight lymphocytic and plasma cell infiltration. The interstitial septa are markedly edematous, hyperemic, broadened and contain some swollen mononuclears, which are occasionally seen as large eosinophilic cells with bean-shaped nuclei inside of the alveolar spaces. The alveolar lining is not conspicuous. (Figure 1.)

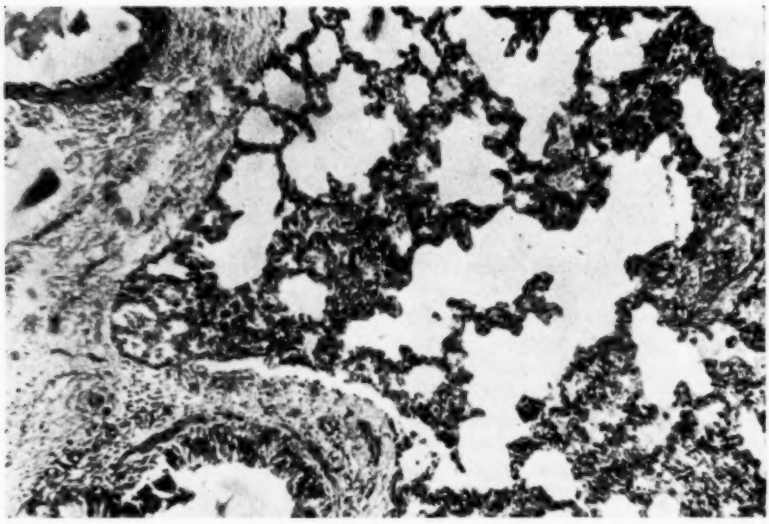

Fig. 1—Congestion, edema, spotty atelectasis and thickened septa of alveoli. Edema of interstices.

The picture is that of so-called interstitial pneumonia or, using a term recently introduced by Gruenwald and Jacobi, mononuclear pneumonia (10). Mononuclear inflammation of the lung without neutrophils or with only a fleeting polymorphonuclear reaction is really the characteristic of a large group of pneumonias of varying etiology. Though morphologically not always distinguishable and variable, and overlapping at different stages, there are often distinct characteristics which may be helpful in etiologic diagnosis. The forensic autopsy may become important from the public health viewpoint, or as in case of psittacosis, may protect health and life of members of the family of the deceased. Many of these pneumonias still defy etiologic classification. Unraveling the morphologic and clinical entities is often the first step toward a reasonable etiologic classification and toward a sound epidemologic approach. Certain features point to more or less specific entities.

Hyaline membranes are common in influenza (8, 13), and primary atypical pneumonia (12), and are sometimes present in psittacosis and psittacosis-like pneumonia (11). They are also seen in pneumonia due to meningo-pneumotropic virus (5). Here they are found often with extensive consolidation

from a severe mononuclear proliferation and exudation uncommon to such extent in other types.

Swollen alveolar cell lining is likewise a characteristic of influenza, primary atypical pneumonia and psittacosis, and was also seen in the severe epidemic of Louisiana pneumonitis (4) caused by Miyagawanella louisianae (5). It presents a marked serofibrino-sanguineous exudate similar to the Rickettsial pneumonia of Q-fever (14).

Freedom from exudate of the bronchioli is seen in primary atypical pneumonia in its pure form. Here, pleurisy is common while absent or minor in the psittacosis group. The latter is characterized by consolidation spreading outward from the hilum. Histologically one gains the impression of a lobar pneumonia with mononuclears instead of leukocytes and with stages varying from field to field of the slide.

Interstitial pneumonia is pronounced in primary atypical pneumonia, in Q-fever, and is also seen to a slight extent in Louisiana pneumonitis, but is inconspicuous in pure influenza.

There is further a group of pneumonias occurring in newborns, infants and young children characterized by inclusion bodies. Intracytoplasmatic inclusions in pneumonia were described by Adams (1) causing nursery epidemics, occasionally with sudden death in premature infants. The acidophilic inclusions, 3 to 6 mu, are surrounded by a halo and occur mainly in bronchial epithelium. *Nuclear* inclusion pneumonia of Goodpasture has likewise caused sudden death in a two-month-old infant. It has herpes-like, granular bodies filling the nucleus of the epithelium from the trachea down to the alveoli. This entity is most often observed in measles and pertussis. There is the giant cell pneumonia of Hecht (15) with both nuclear and cytoplasmatic inclusions, which are round or oval, likewise acidophilic, often multiple measuring 2 to 10 mu in size, and in giant cells up to 25 mu, similar to distemper. They are found in subacute and chronic epidemic pneumonia of infants and young children. Cytomegalic nuclear inclusions (1), basophilic and enlarging the whole cells may affect the lung as they do many other organs, commonly the salivary glands.

Finally a peculiar type of pneumonia (9) has been observed in Europe, first described by Bennecke in 1938 under the name of plasma cell pneumonia, and has become a major problem.

Occurring in premature and debilitated infants, it is also observed in normal babies up to the age of six months, often about the 9th week. It is characterized by a massive, occasionally tumor-like interstitial infiltrate with plasma cells and a peculiar alveolar exudate consisting of a delicate network of eosinophilic fibers with fine (1 mu) basophilic granules. It may be worthwhile to watch for this entity in the United States.

There are numerous problems connected with these rather inconspicuous pulmonary changes we call interstitial pneumonia. The forensic pathologist, above all, is a physician dedicated to human welfare. Death must not be ascribed to suffocation when due to disease. It causes untold anxiety, the social stigma of neglect of one's child, and unjustifiable criminal proceedings. But the duty does not end there. A personal interview with the parents seems to be a rewarding task. Severe grief reactions are due to guilt feelings generally occurring 5 to 6 weeks after the event. I have made it a rule to tell parents to come back to see me if further questions arise in their minds a few weeks later, and then let them talk about what they thought to be their fault. If it does not help I recommend a capable psychotherapist, who in two or three sessions is generally able to relieve their minds.

On the one hand, we cannot forget that infanticide occurs. How do we know disease was a factor in death? Generally the autopsy as a whole will tell. We do not make our diagnosis on single findings, but in summarizing everything we can discover. Signs of infection, common in later life, are lacking in young children. There is no alarm reaction of the adrenal; the lipoid content is unchanged (3). The spleen is rarely swollen and septically softened before the age of seven. The most valuable signs of sudden death without preceding disease, is the glycogen content of liver cells. The granular, glycogen-containing cytoplasm seen in normal liver cells of biopsies should always arouse suspicion when found in an autopsy specimen. Death then occurred in full health. Lymphoid tissue with over-active germinal centers (10), or atrophic lymph follicles with indistinct boundaries in spleen or lymph nodes, and signs of atrophy of the thymus (17), point to disease.

Control cases of hemorrhagic or other shock in babies, of several hours to half a day duration, may give us further insight into the pulmonary changes.

Is interstitial pneumonia a pneumonia? I do not know. Do we create a status lymphaticus redivivus and call it interstitial pneumonia? Do we really see in the lung the primary lesion of death, or merely a response to a multitude of injuries as in the shock kidney?

What is the cause of death? Suffocation from these often rather unimpressive pulmonary lesions? Cyanosis and respiratory distress are surely observed in those infants when seen while still alive.

Is it shock from overwhelming infection? Certain viruses, for example, the influenza virus, are toxic when injected in large amount and cause death (16). Many a virus refuses to multiply except in man and escapes the usual methods of discovery.

Nonobstructive vomitus is a common finding. Do glottis spasm and reflex mechanisms play an important role?

Swelling and edema of the brain, which in children has far less span for expansion than in adults and especially in older people, may play an important role in some of the cases of sudden death (6, 7).

Archibald (2) has pointed to a possible mechanism caused by slight lack of O_2; stimulated pulmonary ventilation, lowered alveolar CO_2, lower arterial CO_2, alkalemia, depressed breathing, cerebral anoxia, loss of consciousness and death. Can certain cases we call interstitial pneumonia be explained in this manner? It is physiologically unsound (18). But, it points to the possibility that a sleeping baby, especially one with a cold, lying under a bed cover or on his face, may rebreathe CO_2. Unconsciousness and death might result without struggle, similar to death from anoxia in high-altitude flying. Further research in this direction is needed. Let us not forget a possibility which may not yet be disproven, and in our effort to avoid injurious diagnoses, let us not miss a fight against a cause of death.

Interstitial pneumonia, so-called, poses many more questions than have been answered. One great lesson we have

learned: Not to accuse parents of neglect, without proof, when disease has taken their baby.

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"The History of American Funeral Directing"

Robt. W. Habenstein and Wm. M. Lamers

Published by Bulfin Printers, 1955, 636 pages, 135 illustrations, including an appendix.

This interesting and informative book represents a monumental amount of information devoted to tracing the obscure origins and development of the modern American Funeral Director. In unfolding the story of the American Funeral Director light is cast on many related customs, laws and procedures relating to funerals, wakes, cemeteries, cremations, embalming materials and methods, caskets, vaults, vehicles, establishments and educational requirements.

The book traces the origin of the funeral director from about 4000 B.C. in Ancient Egypt where the embalmer-priest combined both technical and religious functions, through Greek and Roman culture with their funeral conductors, into the Dark Ages and early Christian-Jewish customs to the Middle Ages where embalming was practiced by physicians in some countries and by barber-surgeons or candlemakers in others. England and its customs are emphasized, as most of colonial American funeral practices are related to English practices. In the Colonies the funeral director was associated with one of two other occupations—cabinet maker or livery stable operator. During the Civil War the greatest increase in interest in the practice of embalming was observed. In the Post Civil War Period is noted the first establishment of regular schools of instruction in embalming technique and with the close of the 19th Century the rise of regular funeral establishments devoted to the care of the dead and the conduct of funeral ceremonies.

The book is well written and thoroughly documented with page annotations and an excellent Bibliography. The Appendix contains a history of the U. S. Army Graves Registration Service, and information concerning various Funeral Directors Associations, Schools of Mortuary Science, and Mortuary Journals.

EDWARD C. JOHNSON

Basal Subarachnoid Hemorrhage and Trauma*

Richard Ford, M.D., Boston, Mass.

Minor subarachnoid bleeding in relation to head injury is common and is accompanied by signs of great violence such as fracture of the skull and contusion or laceration of the brain. Occasionally in the presence of a subdural hematoma the torn dural-cortical bridging vessel may be readily identified by a small area of subarachnoid hemorrhage at the site of its cerebral attachment. Such subarachnoid hemorrhages are small though occasionally multiple and confluent, and frequently occur over the convexities of the cerebral hemispheres.

Massive *basal* subarachnoid bleeding on the other hand commonly results from ruptured congenital aneurysms, either gross or microscopic in size, with extension of bleeding upward on the lateral aspects of the cerebrum and often through the foramina of Magendie and Lushka as far as the third ventricle. Arteriosclerotic and mycotic arterial defects are less frequent sources for basal subarachnoid hemorrhages as are intracerebral hemorrhages which rupture secondarily into the ventricular system and emerge at the fourth ventricular foramina filling the cisterna interpeduncularis. Chronic hypertension is a frequent precursor of arterial rupture. Premonitory signs of arterial rupture may appear in the form of syncope, dizziness, headache for hours or days—in one case six years (Figure 1)—in advance of terminal hemorrhage, or the onset may consist of sudden pain followed by immediate coma.

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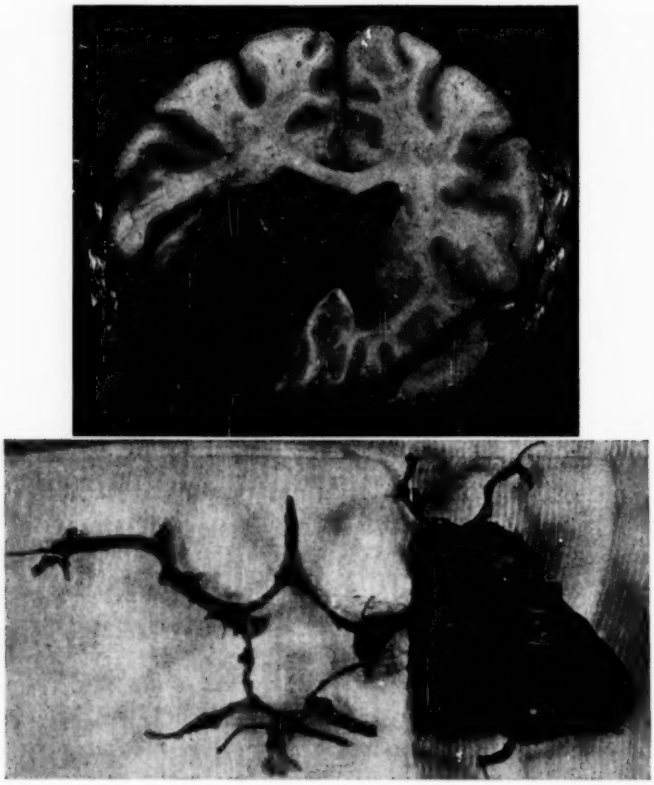

Fig. 1—Intracerebral and subarachnoid hemorrhage following rupture of massive congenital aneurysm of right middle cerebral artery. Diagnosis of "cerebral hemorrhage" six years prior to onset of coma while driving automobile. Laminated thrombus fills major portion of aneurysm.

The majority of cerebral arterial ruptures occur either during sleep or minimal activity without any question of precipitation by stress or trauma. Occasionally, however, civil or criminal proceedings are contemplated because emotional stress, physical exertion, or head injury is said to have aggravated a pre-existing cerebral arterial disease causing rupture and basal subarachnoid hemorrhage. It can safely be said that no cerebral arterial rupture *within* the substance of the brain results from head injury without evidence of disruption of brain tissue by trauma—disruption other than that due to dissecting blood from the rupture. Brain tissue is far more

delicate than even the most thin-walled aneurysm which, weak though it may be, is yet capable of withstanding systolic blood pressure. When arterial defects are located upon the surface of the brain, arterial subarachnoid bleeding may rarely result from rotation of the brain exerting traction upon the circle of Willis (see case 9). This mechanism is similar to the tearing of bridging veins in the production of subdural hematomas (1). *In order to conclude that brain rotation within the skull precipitated the rupture of a basal cerebral artery there must be clear evidence of a severe impact to the head.* The force that commonly contributes to the premature rupture of cerebral arteries is a sudden increase in systolic blood pressure due to physical exertion or emotional stress such as pain, fear, rage or excitement, or to a combination of physical stress and emotion. Numerous determinations of systolic arterial pressure during physical exertion are recorded in medical literature. A systolic rise from 120 mm. Hg to 200 mm. Hg or more is not uncommon during violent effort. The values of systolic arterial pressure during intense emotion are understandably difficult to find. During sexual activity the systolic pressure in man has been reported to rise from 120 mm. Hg to 250 mm. Hg, and in woman from 110 mm. Hg to 200 mm. Hg, with diastolic rises from 80 mm. Hg to 120 mm. Hg and 80 mm. Hg to 105 mm. Hg respectively (2). Cardiovascular and cerebrovascular failure during sexual activity have been reported since ancient times (3). By applying the accepted legal test for the burden of proof for civil action it may be shown *by a preponderance of the evidence* that physical or emotional stress precipitated arterial rupture. In homicide prosecution it is necessary to show that violence directly—not through emotional response alone—set in motion the chain of events leading to arterial rupture, subarachnoid hemorrhage and death. In view of the possibility that the onset of hemorrhage may have been precipitated by the emotion evoked prior to trauma it is usually difficult to assert that *beyond a reasonable doubt* a minor blow is the proximate cause of the death. The medicolegal pathologist who himself fails carefully to evaluate the medical evidence hands to laymen a problem for the solution of which they have neither training nor experience.

The following ten cases gathered over a period of seven years are briefly presented to show a progression of the pre-

cipitation of cerebral arterial rupture by emotion alone, to trauma without emotional component.

Case 1: A 45-year-old mother of seven children collapsed during coitus with her husband and immediately became comatose. She was taken by police ambulance to a hospital and was dead on arrival. At autopsy the sole significant pathologic finding was a subarachnoid hemorrhage (Figure 2) arising from a ruptured congenital aneurysm of the right middle cerebral artery superior to the tip of the temporal lobe. No history of symptoms prior to the fatal hemorrhage was obtained. Although coincidence cannot be completely disregarded, the emotional activity at the moment of onset of symptoms is probably the precipitating cause.

Fig. 2—Subarachnoid hemorrhage rising from base of brain. Rupture of congenital aneurysm in woman during coitus.

Case 2: A 33-year-old married female chronic alcoholic was found dead at noontime in the bed of a male lodger in a rooming house. The man's story was that she was brought to his room on the previous evening by another man who engaged in sexual intercourse with her and then departed. During the night the roomer took advantage of his opportunity and also performed coitus. An undetermined quantity of alcohol was ingested during the night. Examination of the place of death showed no disorder. No injuries were present about the head, face or neck. Over the anterior iliac regions there were two small contusions, and a third was present on the left thigh. The body was fully clothed but the crotch of the underpants was displaced laterally. At autopsy, there was found a mod-

erately fatty liver and a basal subarachnoid hemorrhage. No macroscopic aneurysm was found and there were no atherosclerotic changes of the cerebral arteries. The blood alcohol was 0.40 gm/100 ml. An even greater emotional activity than in case 1 may reasonably be presumed to have occurred.

Case 3: A 34-year-old drunken male entered a tavern and approached a table at which were seated a young man and two young women. He offensively offered his attentions to one of the women and was restrained forcibly by the young man and the bartender pending the arrival of police. Suddenly, in the course of struggling violently, he slumped to the floor unconscious. He was dead on arrival at a hospital. On examination of his body, the only injury was a three-quarter-inch superficial laceration of the dorsum of the left hand. The cause of death was a basal subarachnoid hemorrhage. No gross aneurysm was demonstrated. The blood alcohol was 0.28 gm./100 ml.

Case 4: A 48-year-old male was last seen alive leaving a tavern. One-half hour later he was found dead in a doorway face down on a flight of steps. His sole injury consisted of a laceration of the left lower lip where a tooth was forced through the skin surface (Figure 3). The only significant pathologic finding was a basal subarachnoid hemorrhage, the source of which was not demonstrated. The blood alcohol was 0.20 gm/100 ml.

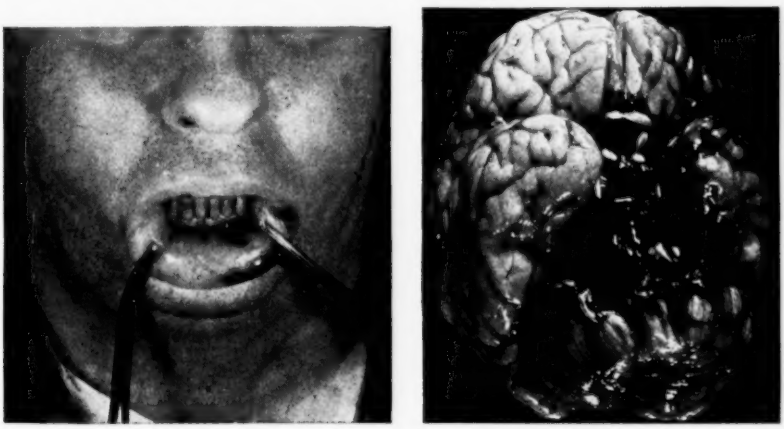

Fig. 3—Contusion and laceration of lip and basal subarachnoid hemorrhage in male found dead with face against flight of steps.

Case 5: Early on a Sunday morning the body of a man was found in an alley. His trousers were down to his knees with the belt and fly still closed; the shorts were below the buttocks and buttoned; and the shirt and suit coat were bunched under the arms and chin. His pockets were ripped open and an empty billfold and personal papers were strewn upon the ground. The only injuries upon his body were longitudinal superficial abrasions over the sacrum and buttocks partly obscured by dirt. The distribution of clothing and abrasions was consistent with the unresisting decedent being half carried, half dragged by two men into the alley for robbery. At autopsy the sole pathologic finding was a basal subarachnoid hemorrhage. No arterial defect was found.

Case 6: A 13-year-old boy was brought to the emergency floor of a hospital where he died within a few minutes. He and a group of chums had been playing with chips of ice at the rear of a delivery truck. A chunk of ice having been dropped inside the neck of his shirt he reacted violently by exchanging punches with the offender and suddenly dropped to the sidewalk in coma. His sole external injury was a small abrasion of the vermillion of the left lower lip. No injury of the scalp was present. The only pathologic finding at autopsy was a basal subarachnoid hemorrhage extending laterally up over both cerebral hemispheres. No obvious source of bleeding was found. It was concluded that the evidence of trauma was negligible and that the precipitating cause was emotion.

Case 7: A 33-year-old male and two companions were seated in a tavern watching baseball on television. Because they engaged in a too-heated argument as to the relative merits of the two Boston teams (both of which were poor that year) they were requested to leave the premises. The verbal exchange was continued in the street until the decedent received a blow in the mouth from one of his companions. He was not knocked down by the blow but collapsed a few moments later. He was dead on arrival at a hospital. The only external evidence of trauma was a trickle of blood from the corner of his mouth. A contusion and one-half-inch laceration were present on the inside of the left lower lip. A severe gingivitis was present and several teeth had been previously lost. The significant pathologic finding was a basal subarachnoid hemorrhage. The arteries of the circle of Willis and

branches were removed and no gross defects were found. The blood alcohol was 0.15 gm/100 ml. It was felt in this case that the injuries to the mouth represented a force insufficient to account for a precipitation of subarachnoid bleeding by trauma. The blow, if it contributed at all, did so by increasing the decedent's rage.

Case 8: A 35-year-old male was one of a group of seven men and one woman celebrating a "birthday party." A quarrel arose concerning priority. The decedent was struck a single blow under the mid-point of the chin. He was said to have hit his head on a washing machine as he fell to the floor. He remained in coma until death in two hours. The only evidence of trauma was a quarter-inch abrasion on the inferior surface of his chin in the mid-line. No contusion of the scalp was present. There was a basal subarachnoid hemorrhage with no discernible arterial defect. The defendant stated that he was obliged to defend himself against a hatchet in the hand of the decedent. Medical opinion was offered that the violent rage of the decedent was a more plausible reason for the subarachnoid hemorrhage than the blow to the chin.

Case 9: A 72-year-old man failed to return home for his Sunday dinner. It was his custom to spend Sunday morning in the dry-cleaning plant owned by his sons, supervising the work of the clean-up man. He was found lying dead in the combination strong room and stock room of the plant with his head propped against two gallon jugs of cleaning fluid and his hands tied with a necktie. The steel door with its combination lock was open. Beside him was a high stool on which was a spattering of dry blood. Beyond him lay his hat. At his feet was a new wet mop head (ruse for opening the strong room) destined for the empty mop handle lying upstairs on the coat of the missing janitor. Gone with the fugitive were the Saturday night receipts from beneath the deposit chutes in the strong room. The position of the body, the blood on the stool, the presence of floor dirt only on those surfaces of the clothes in contact with the floor, and the absence of drag marks on the counters of the new shoes indicated that the old man had gone down and never moved again. Two small abrasions covered with a small amount of dry blood were present on the left jaw and tip of the chin. In

the right occipital scalp was a hematoma with a quarter inch laceration from which a trickle of blood had run down the jugs supporting the head. The occipital laceration was the probable source for the blood stains on the nearby stool (Figure 4).

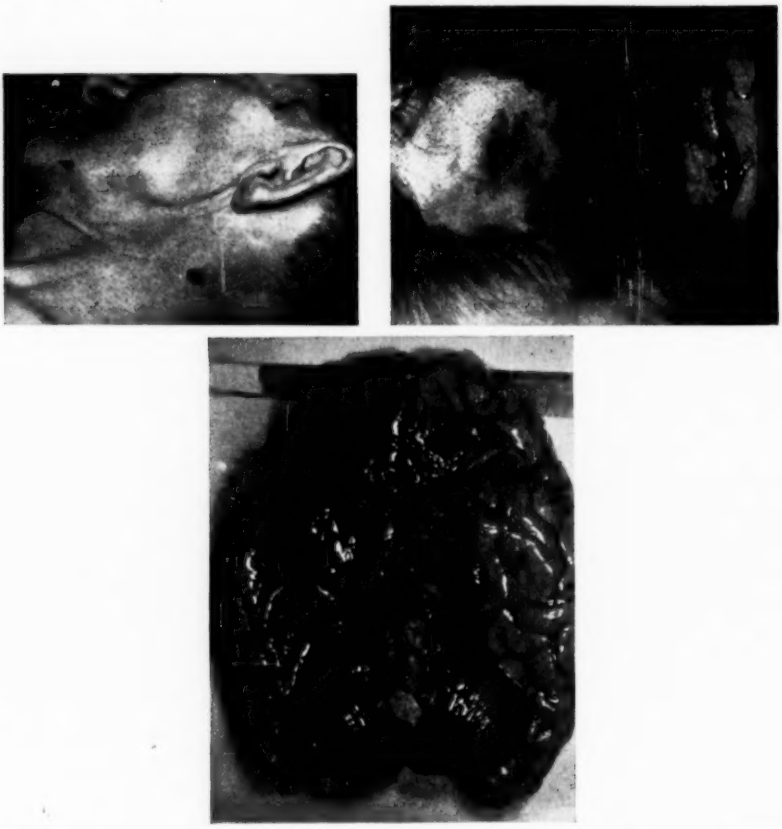

Fig. 4—Abrasions of chin and hematoma and laceration of occipital scalp in man 72 years of age assaulted and hands tied. Senile cerebral atrophy, atherosclerosis, and basal subarachnoid hemorrhage without skull fracture or cerebral contusion.

Post-mortem examination showed generalized atherosclerosis especially marked in the arteries of the circle of Willis, senile cerebral atrophy, and a basal subarachnoid hemorrhage. Neither skull fracture nor cerebral contusions or

lacerations were present. Three ribs were fractured in the right anterior axillary line. In view of the double impacts, chin and scalp, cerebral atrophy—permitting more than usual cerebral rotation, and severe arterial disease—atherosclerosis, it was the opinion of the pathologist that the force applied to the head was the proximate cause of the subarachnoid hemorrhage and of the death.

Case 10: A four-year-old boy and his father were out in a field. The child ran about while the father practiced surf-casting with rod, line and two-ounce lead weight. In the middle of a cast the man saw the child run into the path of the weight and pulled back on the rod to check the flight of the missile. The line, however, snapped down over the boy's head leaving a faint linear abrasion on the right forehead and face. The jointed lead weight, snubbed by the line, struck over the angle of the right mandible and sternocleidomastoid muscle leaving a contusion and abrasion 2¾ by ½ inches in the form of an outline of the weight (Figure 5). The child was immediately unconscious and was dead on arrival at a hospital fifteen minutes later.

Fig. 5—Contusion and abrasion of jaw and neck resulting from impact with surf-casting weight. Death 15 minutes following injury with basal subarachnoid hemorrhage.

At autopsy, there was hemorrhage and contusion of the sternocleidomastoid muscle. The structures of the carotid sheath beneath the site of impact were not injured. Death resulted from a basal subarachnoid hemorrhage without demonstrable arterial defect. No scalp or skull injury was present and there were no contusions of the brain.

In this instance, trauma without any emotional factor appears to be the proximate cause of the subarachnoid hemorrhage. The mechanism for arterial rupture is obscure. Two possibilities exist: Sudden rotation of the head resulting from the father pulling back on the rod and a sudden increase in carotid artery pressure due to direct impact over the carotid sheath. Isolation and sudden compression by a hammer blow of the carotid artery in a dog has so far failed to cause cerebral arterial rupture.

Conclusions: 1. Although in most instances the onset of cerebral arterial rupture occurs during sleep or moderate activity, it may occasionally immediately follow intense emotion, exertion, or trauma.

2. When intense emotion is accompanied by mild trauma the proximate cause of sequential basal subarachnoid hemorrhage is probably the emotion.

3. Evidence of severe trauma to head or neck is necessary to establish injury as the proximate cause of basal subarachnoid hemorrhage in the absence of skull fracture or cerebral contusions.

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